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Nutritional Problems of Children, by Clement A. Smith..... 353
Author Index..... 357
Subject Index..... 365

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NUTRITION REVIEWS

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NUTRITIONAL PROBLEMS OF CHILDREN

A third Diet Conference held at the June, 1961 meeting of the American Medical Association, and sponsored in part by the Nutrition Foundation, concerned problems of current interest requiring diet as a tool in pediatric treatment. Dr. Selma Snyderman of New York University Medical School discussed phenylketonuria. She did so as a well-informed investigator of amino acids in nutrition, with considerable experience in the treatment of children with phenylketonuria. Dr. Snyderman pointed out that beside the genetically determined block in tyrosine production there are also abnormalities of melanin metabolism and of pigmentation of the skin, eczematous eruption, increased sensitivity to epinephrine, and reduced thyroxine formation.

The accumulation of phenylalanine is so rapidly damaging to the brain that little therapy by diet is possible in the established case. But, since subsequent siblings of children with phenylketonuria tend to have the same disease, with abnormally high levels of phenylalanine appearing in the plasma three or four days after birth, treatment by diet can be begun very early in the afflicted siblings of known cases. The problem is not simply one of completely removing phenylalanine, for a certain though variable amount of this amino acid is necessary for growth. Therefore, one first removes essentially all phenylalanine from the diet of a newborn child and then adds small increments until the beginning of weight gain and normal plasma phenylalanine levels indicate that the minimum necessary amount is being given. That amount is usually approximately 30 mg. per kg. per day, as compared with the 180 mg. of phenylalanine per kg. which a normal milk intake would provide. On the administration of such a minimal amount of phenylalanine, electroencephalograms of infants who have already seemed sluggish and of very low intelligence have shown considerable improvement. In infants in whom treatment is begun after the immediate newborn period, the amount of intellectual improvement depends on the age of the child and the amount of damage that has already occurred.

In a sibling of two affected children, abnormal phenylalanine levels in plasma were found at three days of age and the necessary dietary limitation, first to no phenylalanine intake and then to 50 mg. per kg. per day, was begun at that age. The child now tests normally for intelligence at approximately five months. Another child, with treatment begun at nine days, is now 21 months and actually ahead of normal intelligence.

Among numerous interesting details is the fact that cord blood of the affected newborn infant is usually normal since the mother's enzymes substitute for those of the infant. It may be best, therefore, to test the blood of the suspected newborn after feeding a normal diet for a day or two so that the abnormal levels will be more apt to appear. Special foods or complete diets available from commercial firms will lower the phenylalanine intake as much as is necessary. Although these are both unpalatable and expensive, mothers will usually administer them since the alternative is so disturbing. A difficulty to be faced with any infant not intellectually normal is that treatment must be continued at considerable expense to induce even a moderate improvement. It is not known whether the metabolism of some of these children will spontaneously improve; although that hope is a major reason for continued diet therapy.

Phenylketonuria is a rare disease. Among

children registered at well-baby clinics in New York City (many of them Puerto Rican) no cases have been found in the last two years. The incidence is perhaps one child per 100,000 born, and such children may now be discovered before they ever get to well-baby clinics. The eczematous lesions of 20 to 30 per cent of these children offer some opportunity of selection for testing for phenylketonuria, but irreversible cerebral damage may have been done by the time the eczema appears (at three or four months). The possibility of a mild trait in adults, rather than the full-blown disease, is not entirely evaluated. Carriers of the recessive gene may be demonstrated by load tests with phenylalanine. Such individuals convert only a small amount of administered phenylalanine to tyrosine. Their response to the test is between that of the normal individual who shows a marked rise in plasma tyrosine and the phenylketonuric patient who shows no response at all.

Perhaps the most striking result of such work is its stimulus to research upon mental retardation of all kinds, in the hope of finding other metabolic disorders the treatment of which may result in prevention of retardation. Dr. Snyderman mentioned "maple syrup urine disease" as an example, although still rarer than phenylketonuria of this type of disease. One infant with maple syrup urine disease is now under treatment with a completely synthetic diet that limits the intake of the branched chain amino acids.

Dr. M. Rubin described the effect of water restriction and food restriction upon renal function of normal children and noted the elevation of limited urine osmolarity by a high protein diet. He pointed out that in glomerular disease, with the reabsorptive function of the tubule unimpaired and filtration the main function disturbed, dietary treatment may at first consist largely of administration of water in amounts sufficient to cover insensible loss and urine volume. At Children's Hospital in Buffalo, no protein is given to children with severe glomerular failure, and sugar in the form of candy, together with adequate water, make up most of the temporary intake until the azotemia clears. In absence of severe glomerular failure, neither high nor low protein intake affects the ultimate course of the disease. In nephrosis (except the chronic mixed form with glomerular damage also present) changes in body weight and urine volume are carefully observed; the diet contains a reasonable level of protein but is adjusted in water and restricted in salt as indicated by the above evidences of the course of the disease. There is evidence that with more dietary protein (up to 5 g. per kg.), more nitrogen is retained. When cortisone therapy is used, increase of potassium in the diet may be necessary. This increase should be guided by blood samples or, perhaps even better, by electrocardiographic evidence of hypokalemia.

In acute renal shut-down, of whatever cause, reduced filtration of urea, acids, and electrolytes (especially potassium) necessitates avoidance of protein in the diet temporarily, and of such substances as fruit juices which contain rather large amount of potassium. Again, energy is provided by feeding simple sugar in the form of candy but oral or intravenous fat may also be used to spare protein more than carbohydrate alone may do. The danger of careless treatment of severe acidosis with large amounts of sodium-containing solutions, leading to further water retention, and the importance of considering the loss of concentrating capacity, especially in patients scarcely voiding at all, were stressed. On the other hand, Dr. Rubin noted that electrolyte loss and water loss may suddenly become great when much urine is promptly voided at sudden recovery. Again, careful observation of osmolar concentration and electrolytes in the blood serum, as well as

of the patient's weight gain or loss, are fundamentally important in management.

In managing the child with chronic renal failure, congenital or acquired, or with chronic nephrosis, Dr. Rubin advised the physician to discover which functions are lost or more depressed and to deal with these particularly. He pointed out that life can continue for ten years or more, even with glomerular filtration rate reduced to less than 10 per cent of normal. Such situations require reduction of protein intake to the minimum necessary for growth —1 g. or even 0.5 g. per kg. per day—and correction of any tendency to acidosis with oral administration of aluminum hydroxide to cause insoluble phosphates to be formed in the bowel. Also, for the treatment of acidosis in chronic renal failure, Dr. Rubin advises the citrate solution originally introduced by Shohl (100 g. Na citrate, 150 g. citric acid, 1 liter H_2O), 50 ml. per sq. meter per day to replace base lost. Extra water several times daily compensates for failure of concentrating capacity. The use of lactate or bicarbonate solutions was not mentioned. The possibility of underlying renal disturbance in any child failing to grow should be studied by simple measurement of the concentration and acidification of the urine. In disturbances of these functions one may cope to some extent with the renal cause, and thus promote slow if not actually normal growth, through careful regulation of diet.

Dr. A. Kantrow's recommended dietary care of diabetes mellitus in adolescents followed standard practices but with certain interesting additions and comments which stimulated considerable discussion. Dr. Kantrow noted that Fanconi had found a high incidence of nephropathy in diabetic children who had received a low protein diet. Whether this effect is due to less protein in the diet or to excessive carbohydrate or fat is not known. Dr. Kantrow advocated that 15 to 20 per cent of all calories in the diet of adolescent diabetics should be derived from protein. He recommended a water-soluble vitamin supplement (B-complex and C) in addition to a good general diet, for a diabetic adolescent. During periods of rapid growth, Vitamin D may likewise be added. He was questioned by several who doubted especially the need for a vitamin D supplement for children beyond the ordinary rickets age.

The question of the proper food intake of the diabetic adolescent at times of physical exertion has been given a practical answer by those diabetic young men who are champion tennis players and who consume sweetened orange juice before and during tennis matches. It was recommended, therefore, that extra exercise by diabetic adolescents should be preceded or accompanied by extra sugar, which may provide many of the needed calories without increase in insulin. A nondietary item of interest was Dr. Kantrow's use of Orinase to prolong the insulin-free period when an adolescent is first put upon a diet after the discovery of diabetes. Insulin, of course, ultimately becomes necessary. In general, Dr. Kantrow believes that diabetes in the adolescent calls for a fully adequate diet with insulin coverage based on repeated urine tests, rather than for any modification of the diet which could interfere with nutrition or growth.

Dr. Dorothy Andersen's talk on the dietary management of fibrocystic disease aroused perhaps the most discussion of the morning. She noted the consistently and highly acid gastric juice in this disease, as well as the particularly inspissating material in bronchial and other glands; the common factor may be impaired control of salt in the secretion of all glands. Since death when it occurs is usually from pulmonary infection, the question often asked is whether the susceptibility of these patients is a specific result of poor nutrition. It is no longer believed that nutritional factors are primarily responsible for their lowered resistance.

Dr. Andersen stressed the particular importance of diet in the first year or two. Subsequently, prevention of respiratory infection by drugs may become more important and children usually manage to grow if their infection can be controlled. In fibrocystic children between one and five years, balance studies (without added pancreatin) indicate absorption up to 60 per cent of protein and fat and up to some 80 per cent of carbohydrate. Such malabsorption may be compared with absorption of 95 to 100 per cent of the three food substances by normal children.

Dr. Andersen mentioned that soy bean protein is not as well absorbed or utilized for growth by children with fibrocystic disease as are other proteins. In general, two or three times as much protein should be fed fibrocystic children as ordinary children, whereas fat must often be reduced below the level in the usual diet of other children. The diet should not be fat-free; fat-soluble vitamins should be given in high quantity. One of the tests of fibrocystic disease is, of course, the characteristic impairment of vitamin A absorption. Therefore, Dr. Andersen recommends 20,000 units of aqueous (or 40,000 units of oily) vitamin A supplement daily, and the administration of extra vitamin K before any necessary surgery. She believes that these children are also deficient in vitamin E, although this has not seemed to harm them. Water-soluble vitamins are advised on "general principles."

The position of pancreatin as a dietary adjunct for the fibrocystic child is not entirely clear. In Dr. Andersen's experience, no amount of pancreatin entirely replaces all the pancreatic enzymes lacking; balance studies have not clearly indicated evidence of a definite and consistent effect on metabolic balance. However, practice indicates the desirability of giving pancreatin to all younger children with pancreatic insufficiency. During hot weather, unrestricted salt is given by adding about 2 g. in each day's formula for infants and by encouraging free use of the salt shaker by older children. Special dietary problems have arisen in the case of five children with diabetes plus pancreatic fibrosis, and in several others who have pancreatic fibrosis plus cirrhosis of the liver. In general, it seems likely that the anemia often noted in fibrocystic disease is either due to hypovitaminosis K and the resultant tendency to hemorrhage, or to some sort of folic acid deficiency. Dr. Andersen advises the use of folic acid in the treatment of such anemia.

The actual frequency of fibrocystic disease is not known because newborn infants with this disease in severe form often die soon after birth or in early infancy. Dr. Andersen felt that perhaps one in 2,000 children born had pancreatic fibrosis. Pediatricians must therefore deal with this disease considerably more frequently than with phenylketonuria.

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AUTHOR INDEX

The letters SA preceding the page number designate authors of special signed articles; the letter N indicates authors mentioned in the Notes section; LE refers to authors of letters to the Editor; and B designates authors of recent books listed in the Notes section.

Names beginning with Mc are indexed before those beginning with Ma.

Aaes-Jorgensen, E., 62
Abeles, R. H., 85, 283
Abercrombie, M., 44
Abraham, S., 211
Abt, A. F., 278
Acheson, R. M., 135
Achor, R. W. P., 262
Ackerman, R. F., 276
Adair, J., 143
Addis, T., 44, 285
Adelstein, S. J., 4
Adlersberg, D., 325
Aftergood, L., 108, 184
Agar, W. T., 16
Agnew, R. C., 218
Ahrens, E. H., 25
Akedo, H., 16
Akhutin, M. N., 129
Alaupovic, P., 217
Albright, F., 269
Alessio, G. D., 213
Alexander, C. S., N352
Alfin-Slater, R. B., 62, 108, 184
Allcroft, R., 29
Allen, S. I., 206
Allison, N., 42
Allred, J. B., 302
Almquist, H. J., 172
Altland, P. D., 281
Altschul, R., 280, 325
Amos, A. J., B224
Anand, B. K., 202
Andersen, D., 353
Anderson, C. E., 20
Anderson, E., B32
Anderson, E. C., 134, 245
Anderson, J. T., 117
Andersson, M., 257
Andrus, S. B., 88, 281, 306
Anet, E. F., 145
Anichkov, N. N., 161
Anitschkow, N., 221
Arroyave, G., 344
Arteaga, A., 213
Ashburn, L. L., 156
Asper, S. P., Jr., 274
Astrup, T., 37
Auerbach, V. H., 174
Aurora, A. L., 257
Axelrod, A. E., 116, 149, 315
Axelrod, A. R., 274
Babayan, V. K., N127, 275
Bajusz, E., 28
Ball, G. H., 156
Bardawill, C. J., N95
Barker, H. A., 85, 283
Barker, S. B., 174
Barker, W. H., 8
Barnes, L. L., 305
Barnes, R. H., 60
Barritt, D. W., 207
Barry, R. K., 330
Bartholomay, A. F., 4
Basford, R. E., 80
Bauer, G. C. H., 269
Beahrs, O. H., 166
Bean, W. B., 156, 305, 315
Beare, J. L., 184
Beck, A. B., 29
Beck, J. C., 103
Becker, B. J. P., 257
Béhar, M., 324, 344
Bell, M. C., 330
Belliveau, R., 331
Bennetts, H. W., 29
Bentz, J. D., 251
Berg, B. N., 305
Berge, K. W., 209
Bergen, S. S., Jr., 292
Bergman, E. N., 187
Bergstrom, W. H., 152
Berkman, J. I., 85
Berman, D. A., 49
Berman, L., 274
Bernhart, F. W., 82
Berry, H. K., 68
Bertsch, R., 213
Bessey, O. A., N223
Bessman, S. P., 206
Beutler, E., 27
Beveridge, J. M. R., 25, 70
Bickel, H., 68
Biehl, J. P., 8
Bieri, J. G., 52
Bierman, J. M., 129
Bing, R. J., 28
Birnbaum, S. M., 123
Bishop, C. T., 145
Bitot, P., 104
Black, B. M., 166
Black-Schaffer, B., 230
Blackwood, U. B., 148
Blaisdell, R. K., 27
Blalock, A., 179
Blamberg, D. L., 148, 302
Blanc, W. A., 168
Blaxter, K. L., 212
Bleiler, R. E., 293, N352
Blincoe, C., 169
Bliss, A. F., 4
Bloch, K., 113
Bloom, B., 314
Bloom, W. L., N158
Bohra, P., 302
Bomford, R., 274
Bone, J. F., 172
Borinskyaya, E. N., 129
Bosanquet, F. D., 41
Bosley, B., 321
Böttcher, C. J. P., 333
Boucher, R. V., 225
Bourne, G. H., N318
Bower, J. O., 317
Bradley, J. E., 206
Bradley, J. E. S., 232
Bradley, W. B., 88, 186
Bramwell, E. M., 328
Brandt, W. L., 35
Braun, W., 172
Briggs, G. M., 49, 52
Brin, M., 120
Brobeck, J. R., 202
Brock, J. F., B192
Brody, E. A., 200
Bronte-Stewart, B., 25, 207
Brooks, B., 42
Brouha, L., 73
Brown, M. L., 149
Brown, R. R., 8
Brownstein, A. M., 85, 283
Brožek, J., SA129, SA161, B224, 321
Brudevold, F., 124
Brusca, A., 22
Buchanan, J. M., 85, 283
Budnick, L. E., 22
Buescher, R. G., 330
Bunnell, R. H., 146
Burnett, C. H., 242

Burns, J. C., 317
Burns, J. J., 48
Burr, G. O., 17
Burr, M. M., 17
Burrows, B. A., 242
Bush, D. J., 232
Bush, L. J., 77
Buss, E. G., 225
Bustad, L. K., 237
Butler, A. M., 296
Butler, L. C., 79
Byers, J. H., 172
Byers, S. O., 250

Cabot, R. C., 37
Caldwell, E. F., 120
Calhoun, W. K., 88, 186
Calle, G. E. N., B32
Campbell, J. A., 184
Campbell, J. E., 253
Campbell, J. S., N95
Campbell, L., 141
Cantero, A., 44
Carey, J. B., 292
Carlson, R. E., 172
Carlsson, A., 269
Carroll, C. J., 170
Carter, C. W., 156
Carter, R. H., N63
Casey, J. H., 152
Castellanos, H., 344
Caubaud, P., 120
Cava, E. E., 325
Cerecedo, L. R., 60
Chaikoff, I. L., 314
Chalmers, T. M., 39
Chapman, D. M., 338
Chapman, J. E., 267
Chaptman, C. B., 161
Charkey, L. W., 172
Chernick, S. S., 187
Chesney, A. M., 166
Chesrow, F., 2
Chieffi, M., 104
Chiles, T., 151
Chiriboga, C. C., B96
Chow, B. F., 122, 274
Christensen, F., 52
Claborn, H. V., N63
Clark, D. A., 300
Clifton, J. A., N352
Cluett, M. L., N63
Coates, S., 264
Coburn, A. F., N191
Cohen, A. M., 216
Cohen, A. S., 230
Cohen, L., 331
Cohlan, S. Q., 342
Cohn, C., 9, 213
Cohn, I., Jr., 317
Coleman, L. A., 227
Comar, C. L., 42, 78, 245, 308
Combes, M., 195
Combs, G. F., 148, 302
Commons, R. R., 242
Conn, H. O., 206
Connor, W. E., 293
Conrad, H. R., 141
Cook, J., 37
Cook, R. P., 293
Cooperstein, S. J., 80
Copeland, D. H., N160
Corday, E., 209
Cotlar, A. M., 317
Couch, J. R., 302
Counts, M. A., 213
Coursin, D. B., 8
Cox, G. E., 213
Cox, L. G., 213
Craig, J. W., N286
Craig, T. V., 138
Cramer, W., 306
Crampton, E. W., N318
Crane, R. K., 16
Creech, B. G., 52
Creech, O., Jr., 179
Creek, R. D., 302
Crevasse, L. E., 274
Crider, Q. E., 217
Crosby, W. H., N159
Cross, M. J., 85
Crowder, H. M., 42
Cuthbertson, W. F. J., 189

Daft, F. S., 156
Dale, S. L., 262
Dam, H., 25, 52
Daniel, P. M., 41
Darby, W. J., 104, 321, 342
Darling, R. C., 73
Darrow, D. C., 232, 296
Das, M. L., 240
Daughaday, W. H., 1, 216
David, M. M., N95
Davidsson, D., 38, 178, 232
Davies, D. F., 207
Davies, J. N. P., 257
Davies, P., N158
Davis, P. N., 302
Davison, J. R., 170
Day, H. G., 340
Day, P. L., 154, 227
Day, R. D., 269
De Bakey, M. E., 338
de Haas, N350
Deane, H., 44
deCrinis, K., 325
Dehnel, M. K., 145
Dehority, B. A., 212
Delany, K., 60
Dell, J. C., 141
Deshpande, P. D., 186
Deuel, H. J., Jr., 108, 184
Dick, E. C., 251
Dickerson, J. W. T., 247
Dillenberg, H. O., 145
Diller, E. R., 184
Dinning, J. S., 85, 154
Diven, R. H., 172
Dobák, E., 242
Dole, V. P., 289
Douglass, R. C., 274
Drell, W., 156
Drucker, W. R., N286
Drury, D. R., 35
Drury, H. F., 73
Dua, S., 202
Dubos, R. J., 12
Duboucher, G., N191
Dunn, M. S., 156
Dunning, J. M., 259
Dustin, J. P., 68
Dutt, B., 29
Dye, W. B., 169
Dyggve, H., 75

Edwards, D. C., 293
Edwards, H. M., Jr., 302
Edwards, P. C., B32
Eeg-Larsen, N., 42
Eggenberger, H. J., 166
Eggerer, H., 85
Eichel, B., 80
Eijkman, C., 225
Eilert, M. L., 331
Eisenstein, A. B., 22
Ekles, G. H., 344
Elliot, J. M., 170
Ellison, E. H., 138
Elman, R., 285
Elvehjem, C. A., 117, 186, 336
Emmel, A. F., 168
Engel, R. W., 20
Engels, F., 129
Epstein, A. N., 182
Erdmann-Oehlecker, S., 200
Ershoff, B. H., 17, 309
Erwin, E. S., 344
Estren, S., 200

Etteldorf, J. N., 296
Evans, H. M., 17
Evans, J. M., 207
Evans, W., 268

Faloon, W. W., 72
Fanconi, V. G., 135
Faragalla, F. F., 88
Farber, E., 118
Farnan, P., 230
Fawcett, D., 44
Fedoseev, A. N., 346
Fee, B., 266
Fell, H. B., 91
Ferguson, T. M., 302
Fiala, G., 60
Fillios, L. C., 281
Fineberg, S. K., 202
Finerty, J. C., 17
Fisher, A., 120
Fisher, B., 315
Fisher, H., 244
Fitzgerald, R. J., 105
Flett, J., Jr., 296
Flinn, J. H., 325
Fontanares, P. E., 278
Forbes, G. B., 134
Forbes, R. M., 178
Ford, F. J., 324
Fottrell, P., 344
Fowler, D., 68
Francis, G., 145
Frape, D. L., 91
Fraser, R., 269
Fregly, M. J., 57
Freibrun, J. L., 209
Frenk, S., 232
Fricke, H. H., 48
Fridhandler, L., 16
Frieden, E., 174
Friedman, D. L., 85
Friedman, L., 146
Friend, C. J., 344
Friend, D. W., N318
Friendberg, C. K., 342
Friis-Hansén, B. J., 232
Fritz, I. B., N286
Frolov, J. P., 161
Fry, G. S., 20

Gabin, G., 20
Gaertner, R. A., 179
Gaffney, G. W., 122
Galdston, I., B256
Gallup, J., 134
Gallup, W. D., 172
Galton, V. A., 174
Ganelina, I. E., 161
Ganguly, J., 344
Garbers, C. F., 344
Gardner, D. E., 124
Garn, S. M., 36
Garner, R. J., 237
Gáti, B., 242
Gavosto, F., 22
Gelfand, D. P., 129
Gel'shtein, E. M., 161
Gershoff, S. N., 88, 306, SA321
Ghadimi, H., 68
Gibbs, M. H., 251
Gibson, J. G., 4
Gilfillan, J. L., 251
Gillman, J., 344
Glass, H. B., 80
Glick, D., 79
Goldbloom, R. B., 168
Goldner, M. G., 289
Gómez, F., 232
Goode, R., B32
Goodhart, R. S., 4, 129
Goodpasture, E. W., 37
Gopalan, C., 232
Gorchakov, A. K., 129
Gordillo, G., 232
Gordon, B. S., 2
Gordon, R. S., 52
Gorham, P. R., 145
Gornall, A. G., N95
Gotsis, A., 117
Gould, R. G., 113, 314
Grainger, R. M., 311
Grande, F., 117
Graves, R. R., 170
Green, D. E., 20, 80
Greenberg, S. M., 17
Greenstein, J. P., 123
Gresham, G. A., 333
Grice, H. C., 184
Griffiths, D., 105, 311
Grifo, A. P., Jr., 172
Grummer, R. H., 4
Gubner, R., 289
Guerrant, N. B., 60
Guest, J. R., 85
Guha, B. C., LE31, 240
Gullino, P., 342
Gurian, H., 325

Haddow, A., 195
Haig, C., 4
Haley, M. I., 100
Hall, P. F., 257
Halpert, B., 338
Hamada, G. H., 245, 308
Hammarsten, G., 88, 306
Hand, D. B., 227
Hannon, J. P., 73
Hansard, S. L., 42
Hardinge, M. G., 70, 309
Hardy, E. P., 245
Hargis, B. J., 315
Harland, W. A., 216
Harley, R., 29
Harper, A. E., 20, 117, 186
Harpur, E. R., N127
Harris, P. L., 146
Harris, R. S., 314
Harrison, H. C., 42
Harrison, H. E., 42
Harrison, M., 269
Harrison, R. G., 218
Hartmann, A. F., 296
Hartree, E. F., 80
Hartroft, W. S., 331
Hartz, R., 82
Harvey, O. A., 184
Hashim, S. A., 213, 292
Haskell, J. A., 36
Hastings, A. B., 272
Hatch, F. T., 283
Hathaway, M. L., SA195
Haubrich, W. S., 230
Hays, R. P., 300
Heaton, F. W., 218
Hegsted, D. M., 25, 37, 92, 117, 178, 225
Heinrich, H. C., 200
Helgebostad, A., 143
Helleiner, C. W., 85
Hellerstein, E. E., 92
Hellerstein, S., 232
Henchel, A., 161
Henderson, L. M., 186
Hendrix, T. R., 230
Hennessey, D. J., 60
Hepburn, F. N., 186
Herndon, J. F., 49
Heroux, O., N95
Hetler, R. A., 104
Hibbs, J. W., 141
Higginson, J., 257
Highman, B., 281
Hilbish, T. F., 198
Hildebrand, J. E., 17
Hillman, R. S. L., 16
Hoch, F. L., 4
Hodge, H. C., 139
Hodges, R. E., 156, 293, 315
Hodgkinson, A., 306
Hodgson, R. E., 170
Hoeber, J., 16

Hoeber, R., 16
Hoefer, J. A., 172
Hoekstra, W. G., 4
Hoerlein, A. B., 77
Hogan, J. P., 187
Hogenkamp, H. P. C., 85
Holiday, M., 232
Hollander, W., 242
Holliday, M. A., 242
Hollinshead, B. S., B224
Holloway, P. J., 311
Holman, R. T., 62, N127
Holmes, W. L., 251
Holt, P. R., 230
Hopkins, C. Y., 184
Hopper, S., 116, 315
Hoppert, C. A., 105, 311
Horning, E. S., 195
Hornung, H., 259
Hove, E. L., 20, 49, 146
Howard, A. N., 333
Howell, W. L., 37
Hsia, D. Y., 234
Hsu, J. M., 122
Hueper, W. C., 227
Huff, J. W., 251
Huffman, C. F., 170
Huisman, T. H. J., 68
Hungate, R. E., 170
Hunt, A. D., Jr., 229
Hunt, H. R., 105, 311
Hunt, J., 23
Hurley, N. A., N223
Hursh, J. B., 134
Hutt, F. B., SA225

Il'inskii, B. V., 161
Ingbar, S. H., 174
Irving, D. W., 209
Isenberg, H., 85
Issekutz, B., Jr., 115
Isselbacher, K. J., 230
Ivy, A. C., 166, 309

Jacob, F. A., 16
Jacobson, E. D., 72
Jacobson, J., 213
Jacobson, S. D., 274
Jacquot, R., B32
Jaffe, H., 209
James, J., 195
Jankelson, O. M., 37
Jarnum, S., 236
Jenkins, K. J., 42
Jennings, R. B., 88
Johnson, B. C., 14, 49, 60, 110, 156, 217
Johnson, D., 113
Johnson, R. E., 73, N127, 275
Jones, C., N63
Jones, H. G., 237
Jones, I. R., 172
Jordan, D. C., 77
Jordan, G. L., Jr., 338
Joseph, D., 9
Joslin, E. P., 216

Kabler, J. D., 138
Kagan, A., 328
Kagi, J. H. R., 4, 340
Kalant, N., 216
Kalinsky, H., 120
Kano, A., 172
Kantrow, A., 353
Karvinen, E., 309
Katz, L. N., 213
Kaunitz, H., N127, 275
Kawahara, H., 63
Kayahan, S., 90
Keilin, D., 4, 80
Keith, R. A., 100
Kekwick, A., 39
Kempner, W., 35
Kennedy, G. C., 247
Kerner, I., 168
Kerpel-Fronius, E., 242
Keyes, P. H., 105
Keys, A., 117, 151, 155, 161, 294, 309
Khalatov, S. S., 161
Kidder, G. W., N318
Kifer, P. F., 311
Kik, M. C., 175
Kim, S. M., 309
Kimball, O. P., 166
Kimble, M. S., 101
King, C. G., 48
Kinsell, L. W., 25
Kipschidze, N. N., 221
Kirk, J. E., 104
Kirnsky, N. I., 344
Kirschner, S. L., 314
Kisliuk, R. L., 85, 283
Klain, G. J., 49
Klein, A. K., 340
Klein, G. F., 17
Klein, P. D., 184
Klein, S., 245
Knight, P. L., 60
Knox, W. E., 174, 234
Kobernick, S. D., 55
Kodicek, E., 240
Koenig, E., 129
Kokatnur, M., 113
Konishi, F., 42
Kopland, D. V., 170
Kornberg, A., 156
Korzenovsky, M., 184
Kotake, Y., 336
Kraepelin, E., 274
Kratzer, F. H., 302
Krause, M. V., B320
Krauss, W. E., 141
Krautmann, B. A., 49
Krehl, W. A., 79
Krishnamurthy, S., 344
Kulczycki, L. L., 88
Kummerow, F. A., 113
Kwaan, H. C., 37
Kwong, E., 60

Ladd, J. N., 85
Lagacé, A., 169
Landgon, R. G., 113
Langham, W. H., 245
Larson, F. C., 8
Larsson, S., 202
Lasser, R. P., 342
Lát, G., 247
Laurie, W., 257
Lavrov, B. A., 129, 161
Lawrence, J. S., N352
Lawson, J. D., 274
Lazarus, S. S., N254
Leach, E. H., N96
Leary, T., 37
Lease, J. C., 111
Lee, H. A., Jr., 85
Lee, J. M., 315
Lee, W. A., 124
Leftwich, W. B., 116
Lehman, A. J., N63
Lehmann, H., 42, 78
Leitner, Z. A., 101
Lemley-Stone, J., 325
Lengemann, F. W., 42, 78
Leone, N. C., 198
Lepkovsky, S., 17
Leverton, R. M., 6, B32
Levine, R., N286
Lew, W., 44
Lewis, H. B., 16
Lewis, P. K., Jr., 4
Lightwood, R., 135
Lin, T. M., 309
Lindner, H. R., 187
Lindquist, B., 269
Lindsay, R. H., 174
Ling, C. T., 122
Linnér, E., N96
Lipton, M. A., 336

Lisan, P., 262
Liu, C. H., 42
Livingstone, P. D., N63
Lloyd, J. P. F., N96
Logue, R. B., 274
Long, D. A., 315
Longenecker, H. E., 48
Loosli, J. K., 170
Lorie, I. F., 129
Lough, S. A., 245, 308
Lowe, J. S., 218
Ludwig, M. I., 146
Lund, C. J., 195
Lunin, N. I., 161
Lurie, G. M., 324
Lynen, F., 85

McAleese, D. M., 178
McAllister, F. F., 213
McCance, R. A., 42, 78, B128, 247
McCandless, E. L., 276
McCarrison, R., 257
McCay, C. M., 42, 305
McClure, F. J., 124
McClure, F. M., 198
McClure, R. D., 166
McConnel, K. P., 330
McCoord, A. B., 168
McDonald, M. W., 225
McElroy, W. D., 80
McFadzean, A. J. S., 37
McGrath; H., 178
McHenry, E. W., 20, 120
McLaren, D. S., 104
McLarenhune, M., 1
McNally, K. S., 77
MacDonald, A. M., 212
MacDonald, R. A., 83, N160
MacFarlane, R. G., 156
Machlin, L. J., 52
MacIntyre, I., 38, 178, 232
Mackler, B., 80
Macy, I. G., 308
Maddox, G., B32
Mahavdevan, S., 344
Makarychev, A. I., 129
Malkiel, S., 315
Mallory, G. K., 83
Malm, O. J., 42
Mameesh, M. S., 14, 60, 110
Mandel, E. E., 37
Manion, W. C., 37
Manley, R. S., 139
Mann, T., 4
Mannering, G. J., 336
Marcus, S. C., 166
Marine, D., 166
Marsh, C. L., 77
Marsh, E. M., 120
Marsh, M. E., 331
Marsh, P. L., 216
Martin, A. J. P., 168
Martinsons, E., 143
Marvin, H. N., 154
Mashford, M. L., 207
Mason, K. E., 17, 146, 168
Mason, R., 73
Massari, F. S., 317
Mattern, C. F. T., 85
Matterson, L. D., 189
Mattson, F. H., 62
Maw, A. J. G., 225
Mayer, A. L., 88
Mayer, J., 182, 218
Meadows, R. W., 2
Mehler, A. H., 174
Meijer, W., B352
Meisky, K. H., 52
Melby, J. C., 262
Mellanby, E., 91
Mellinkoff, S. M., N352
Mendes, C. B., 272
Meneely, G. R., 342
Mengle, H. A., 317
Merezhinskii, M. F., 161
Merrill, J. M., 325
Mertz, W., 348
Metcoff, J., 232, 272
Metta, V. C., 14, 110
Meyer, J. H., 342
Mezhebovskii, R. G., 161
Mickelsen, O., 23, 47, 161
Mikuta, E. T., 174
Miller, A., 200
Miller, A. R., Jr., 202
Miller, B. F., B32
Miller, H. C., 266
Miller, J. M., 299
Miller, M., N286
Mills, C. F., 29
Milmore, B. K., 166
Mirick, G. S., 116
Mitchell, J. R. A., 207
Mitchell, R. G., 135
Modell, W., 202
Molchanova, O. P., 129, 161
Mollin, D. L., 200
Molony, C. J., 8
Monroe, C. F., 141
Montemurro, D. G., 155
Montgomery, R. D., 38
Monty, K. J., 85
Moodie, A., 324
Moore, R. E., N255
Moore, T., N96, 101, 168, 218, N318
Moreno, A. H., 300
Morgan, A. F., 79
Morgan, A. L., 104
Morino, Y., 336
Morris, J. N., 328
Morrison, A. B., 111
Morse, E. H., 70
Morton, R. A., 218
Moses, C., 179
Moss, W. G., 179
Moyer, J. H., 262
Muckell, J., N288
Mueller, J. F., 8
Mueller, P. S., 137
Mulford, K. E., 227
Mullan, B., 269
Mullins, A. G., 211
Muntz, J. A., 272
Murphy, E. A., 17
Murphy, G. T., 120
Murray, G., N222
Murray, T. K., 184
Musulin, R. R., 48
Muth, O. H., 41, 169
Myasnikov, A. L., 161, 221

Nagoya, N63
Naito, C., 281
Nakada, H. I., 88
Nakahara, I., 336
Nakamura, M., 92
Nakatsu, S., 49
Nath, N., 117
Necheles, H., 151
Nelson, D. A., 88
Nelson, J. B., 12
Nestel, P. J., 207
Neufeld, A. H., 100
Newbrun, E., 124
Newburgh, L. H., 216
Newell, G. W., 139
Newman, H. A. I., 276
Nezamis, J. E., 151
Nichol, C. A., 22
Nicol, B. M., 104
Nicolaev, O. V., 129
Nicolaysen, R., 42
Nicholson, J. W. G., 170
Nishida, T., 113
Niwayama, G., 55
Nold, M. M., 42, 78
Nordin, B. E. C., 269
Nordsieck, M., 211
Norman, A. P., 68, 264

Normand, I. C. S., N255
Novikoff, A. B., 44

Oaks, W., 262
O'Brien, D., 68
O'Brien, J. R. P., 156
O'Dell, B. L., 49
Odell, G. V., 77
Oesterling, M. J., 186
Oettle, A. G., 257
Ogawa, T., 151
Okamoto, M., 212
Okey, R., 213
Okuda, K., 274
Okunev, N. V., 161
Okunuki, K., 80
Oldfield, J. E., 41, 143, 169
Olitsky, P. K., 315
Olson, J. A., 4, 334
Olson, R. E., 70, 120, 325
Olsson, N., 225
O'Neal, R. M., 216, 331
Oomen, H. A. P. C., LE350
Opatskii, L. V., 129
Opdyke, D. F., 25
Orland, F. J., 105
Osler, M., 266
Ostashever, A. S., 120
Ott, W. H., 25
Overath, P., 85
Overley, V. A., 70

Page, H. M., 344
Page, I. H., 117
Paine, R. S., 234
Palmer, L. S., 344
Panos, T. C., 17
Parent, R. C., 170
Pařízek, J., 340
Parker, W. H., 29
Parmelee, A. H., 8
Parry, H. B., 41
Parsons, W. B., Jr., 325
Partington, M. W., 264
Patek, A. J., Jr., 4
Paton, D., 104
Patterson, W. I., N63
Paul, J., 344
Pavlov, I. P., 129
Pawan, G. L. S., 39
Pawlowski, M. D., 117
Payne, B. C., N352
Paynter, K. J., 311
Pedersen, J., 266
Peifer, J. J., N127
Peisach, M., 344
Pelc, S. R., 91
Pemberton, J. de J., 166
Pendergrast, W. J., 166
Penrose, L. S., 264
Pepler, W. J., 257
Perez-Santiago, E., N159
Perry, H. O., 262
Person, P., 80
Petersen, V. P., 236
Peterson, J. E., 100
Peterson, M. S., B224
Petrovskii, K. S., 129
Pevzner, M. I., 129
Phillips, P. H., 4, 20, 42, 139
Phillips, W. E. J., 218
Pick, R., 213
Pijanowski, W. J., 299
Pike, R. L., 149
Pileri, A., 22
Pinkos, J. A., 42
Pitney, W. R., 200
Pluckthun, H., 68
Plummer, H. S., 166
Polachek, A. A., 71, 299
Pollack, H., 104, 289
Pollak, L., 78
Pollard, C. J., 52
Popper, H., 2
Posner, I., 53
Poulton, B. R., 141
Pounden, W. D., 141
Prabhu, M. B., 4
Pratt, E. L., 296
Presnell, A. K., 156
Price, J. M., 8
Prien, E. L., 88
Prior, J. T., 72
Pudelkewicz, C., 156
Pudelkiewicz, W. J., 146, 189
Puite, R. H., 230
Pye, O. F., 278
Pyke, W. E., 172

Quackenbush, F. W., 117
Quastel, J. H., 16
Quinn, K. V., 234

Raab, W., 55
Rabinovici, N., N319
Radwin, H. M., 123
Rahman, M. M., 302
Rajagopal, K. R., 218
Ramalingaswami, V., 257
Ramasarma, G. B., 186
Ramirez, R. E., B32
Rand, N. T., 113
Ranke, B., 122
Ranke, E., 122
Rapoport, A., 232
Ratnoff, O. D., 37
Raven, A. M., 78
Rechcigl, M., Jr., 123
Redisch, W., 325
Reed, R., 6, 213
Reid, B. L., 302
Reid, R. L., 187
Reifenstein, E. C., 269
Reinhardt, W. O., 314
Reiser, R., 108
Reisler, A. V., 129
Relman, A. S., 242
Remmert, L. F., 41, 169
Rennels, E. G., 17
Reyes, E., 202
Reyniers, J. A., 60
Richmond, H. G., 195
Richter, C. P., 57
Riddell, C., 293
Rivera, J., 245
Robb-Smith, A. H. T., 156
Robert, A., 151
Roberts, N. R., 22
Robertson, E. C., 104
Robertson, I., 324
Robertson, R. H., 302
Robin, E. D., 4
Robinson, E. E., B32
Rodahl, K., N318
Roderuck, C., 156
Rodger, F. C., N96
Roe, J. H., 278
Roen, P., 209
Roginski, E. E., 348
Rohland, R., 242
Romhányi, G., 242
Rook, J. A. F., 212
Rose, W. C., 186
Rosen, F., 22
Rosen, S., 105
Rosenthal, W., 37
Roth, H. C., 37
Rozental, I. S., 161
Rubin, A. L., 342
Rubin, C. E., N191
Rubin, M., 353
Rubini, M. E., N159
Rubino, F., 21
Rubinov, I. S., 129
Ruegamer, W. R., 250
Ruskin, A., 262
Russell, R. S., 245
Ruthven, C. R. J., N255
Ryss, S. M., 161

Sabiston, D. C., Jr., 179
Sachar, L. A., 285
Sai, F. T., N191
Saiduzzafar, H., N96
Saifer, A., N254
Sakamoto, Y., 336
Sakuragi, T., 336
Salmon, W. D., LE160
Sampson, J., 187
Sandler, M., N255
Sansom, B. F., 237
Sapirstein, L. A., 35
Sarett, H. P., 112
Sarrouy, Ch., N191
Sass, M., 120
Savage, J. E., 49
Sawitsky, A., 85
Schaedler, R. W., 12
Schaffner, F., 2
Schaible, P. T., 302
Schanderl, H., N191
Schedl, H. P., N352
Scheidy, S. F., 77
Scheier, G., 213
Scheinberg, I. H., 133
Schimmel, E. M., 230
Schinazi, L. A., 156
Schleissner, L. A., 209
Schlenk, F., 120
Schmidt, H. J., 139
Schneider, H. A., 315
Schoefield, F. A., 16
Schoenfeld, M. R., 342
Schor, J. M., 174
Schrier, K., 68
Schroeder, H. A., 132
Schroeder, M. A., 331
Schubert, J. R., 41, 169
Schuch, R. L., 134
Schulman, I., 195
Schultz, R. B., 79
Schwabe, A. D., N352
Schwartz, W. B., 242
Schwarz, K., 20, 348
Schwenk, E., 276
Schwimmer, D., 145
Schwimmer, M., 145
Scott, H. M., 49, 113
Scow, R. O., 187
Scrimshaw, N. S., 225, 324, 344
Scriver, C. R., 229
Sebrell, W. H., 156, B192, 292
Sechenov, I. M., 129, 161
Sedov, K. R., 161
Segall, S., 100
Seidel, J. C., 20
Seifter, E., 85
Sellers, A. F., 187
Selye, H., 28
Semashko, N. A., 129
Sendra, L., N191
Senn, M. J. E., 296
Seranova, A. I., 161
Shank, R. E., 44, SA289, 321
Sharman, I. M., 101, 218
Shaternikov, M. N., 161
Shaw, J. H., 105, 198, 311
Shay, H., 151
Sheehy, T. W., N159, LE191
Sheppard, A. J., 156
Shimp, N. F., 80
Shock, N. W., B192
Shore, M. L., 276
Shute, E. V., LE63
Schwachman, H., 68
Sidransky, H., 118
Sie-Boeh-Lian, N350
Sieracki, J. C., 230
Silverman, F. R., 250
Simms, H. S., 305
Simoons, F. J., B320
Singsen, E. P., 189
Sisson, T. R. C., 195
Sitarz, A. L., 195
Skidmore, B. E., 340
Slanetz, C. A., N127, 275
Smart, T. A., 280
Smith, B. B., 187
Smith, C. A., SA353
Smith, F. A., 124
Smith, H. L., 296
Smith, I., 39
Smith, M. G., 285
Smith, R., 232, 269
Smith, R. M., 85
Snell, E. E., 49
Snyderman, S. E., 8, 353
Sodi-Pallares, D., 35
Sorrels, M. F., 108
Sosman, M. C., 198
Spector, H., B224
Sperling, G., 305
Spitzer, J. J., 115
Spivey Fox, M. R., 52
Spurrell, F. A., 187
Srikantia, S. G., 232
Stadtman, E. R., 85
Stamler, J., 113
Stamp, J. T., 41
Standard, K. L., 232
Staple, E., N95
Stapleton, T., 232
Stare, F. J., 70, 117, 309
Starr, P., 209
St. Cyr, M., 262
Steele, J. M., 60, 325
Steiner, A., 179
Stern, J. R., 85
Sternlieb, I., 133
Stetten, D., 39
Stevens, D. F., 276
Stevenson, C. A., 198
Stevenson, J. A. F., 155
Stewart, R. A., N223
Stone, D. B., SA1
Stone, J. B., 170
Stone, S. M., 342
Stout, F. M., 143
Stowe, F. R., Jr., 202
Strauss, E. W., 177
Strominger, J. L., 48
Stunkard, A., 1
Suda, M., 16
Sugawa, T., 16
Sullivan, T. W., 302
Supplee, W. C., 148, 302
Sutro, C. J., 139
Suttie, J. W., 139
Sutton, T. S., 141
Sweeley, C. C., 276
Sweeney, E. A., 311

Tai, M., 120
Takenaka, F., 113
Takeyama, S., 85, 283
Talbot, N. B., 296
Tareev, E. M., 161
Tavormina, P. A., 251
Taylor, C. B., 213
Taylor, C. M., 278
Taylor, H. L., 73, 161
Tchazov, E. I., 221
ten Bokkel Huinink, H., 300
Tennent, D. M., 25, 292
Tepperman, J., 202
Tesluk H., 230
Thomas, J. W., 212
Thomas, W. A., 216, 331, 333
Thomson, J. F., 174
Thomson, J. G., 257
Timiryazev, K. A., 129
Tolgyes, S., N63
Tomarelli, R. M., 82
Tomkins, G. M., 113
Toohey, J., 85, 283
Torrance, E. P., 73
Touster, O., 48
Trowell, H. C., 257
Trujillo, T. T., 134
Trulson, M. F., 6
Tucker, R. G., 342

Tully, R. H., III, 48
Tulpule, P. G., 122
Tumanovskii, M. N., 161
Tunbridge, R. E., 1
Turner, C. W., 141
Tuttle, W. W., 22
Twedt, R. M., 79

Underwood, E. J., 111
Underwood, M. C., N255

Vallee, B. L., 4, 340
van de Kamer, J. H., 300
Van Itallie, T. B., 213, 292
Van Koetsveld, E. E., 29
Van Pilsum, J. F., 49
Vander Wende, C., 80
Varandani, P. T., 91
Varnell, T. R., 344
Vaughan, D. A., 73
Venkatachalam, P. S., 232
Vilter, R. W., 8
Vitale, J. J., 37, 92, 178
Viteri, F., 344
Vogel, F. S., 11
Volcani, B. E., 49
Volk, B. W., N254
Volkova, K. G., 161
Von Hofe, F. H., 195
Von Korff, R. W., 272
Von Schuching, S., 278
Voris, L., SA97

Wacker, W. E. C., 4
Wada, H., 336
Wainio, W. W., 80
Waldbott, G. L., 259
Waldow, A., 207
Waldron, A. M., N352
Walker, A. R. P., SA257
Walker, G. R., 70
Wallace, W. M., 152, 232
Wallerstein, R. O., 195
Ward, G. M., 77
Wasserman, L. R., 200
Wasserman, R. H., 42, 78, 245
Watanabe, Y., 336
Waterlow, J. C., 232, 272
Watkin, D. M., 137
Watson, A. R., 198
Watson, J. H. L., 230
Watson, R. N., 2
Wayne, L. J., 315
Weaver, W., SA65
Weber, G., 44, N127
Webster, B., 166
Week, E. F., 146
Weil, R., 39
Weil, W. B., 266
Weinbren, B. M., N31
Weiner, M., 325
Weinhouse, S., 88
Weinstein, I. B., 200
Weiss, S., 28
Weissbach, H., 85
Weissbein, A. S., 274
Wells, A. F., 108, 184, 309
Wells, O. V., SA33
Wells, R. W., N63
Wells, S., 161
Wespi, H. J., 166
West, L. C., 237
Westmark, G. W., 174
Weswig, P. H., 172
Weyers, H. A., 300
Whelen, W. S., 257
Whereat, A. F., N95
Whipple, G. H., 230
Whiteside, C. H., 302
Whiting, M. G., 6
Wichmann, H. J., 340
Widdowson, E. M., 42, 78, B128, 247
Wiegand, R. G., 8
Wiehl, D. G., 6
Wiener, E., N319
Wiener, R., 117
Wilbur, R. D., 47
Wilcox, A. A., 100
Wilgram, G. F., 331
Wilkins, R. W., 28
Williams, G., 292
Williams, H. L., 8
Williams, J. N., Jr., 20, 122, 285
Williams, M. C., 108
Williams, R. R., B224
Williard, R. F., 71
Willis, P. S., N288
Wills, V. G., 232
Wilson, D., 344
Wilson, P. W., 240
Wilson, T. H., 16, 177
Winitz, M., 123
Winkelmann, R. K., 262
Winters, R. W., 79
Wiseman, G., 16
Wissler, R. W., 331
Wiswell, J. G., 274
Witkop, C. J., 311
Wohl, M. G., 4, 129
Wolf, G., 91, 218
Wolff, J. A., 195
Wolflisberg, H. J., N288
Wolin, E. A., 49
Womack, M., 186
Woodrow, E. P., 324
Woods, D. D., 85
Woods, J. D., 257
Woodward, H., Jr., N286
Woodward, K. T., 134
Woolf, L. I., 68, 264
Worrell, A. C., B32
Wostmann, B. S., 60
Wright, S. W., 168, 264
Wroblewski, F., 120
Wyman, L. C., 315

Yang, M. G., 77
Yeh, S. D. J., 122
Yoon, S. H., 124
Yoshikawa, S., 16
Young, C. M., 6
Young, D. R., 22
Yourish, N. B., 104
Yudkin, J., 221

Zetterqvist, H., 230
Zieve, L., N352
Zilversmit, D. V., 276
Zimmermann, B., 152
Zipkin, I., 124
Zollinger, R. M., 138

SUBJECT INDEX

The letters SA preceding the page number designate a special signed article; N refers to articles or letters in the Notes section.

Abortion
prevention by vitamin E, N63
Absorption
amino acids, mechanism of, 16
calcium
effect of exercise on, 42
effect of lysine on, 78
in osteoporosis, 269
changes, from neomycin, 72
cholesterol, effect of pectin on, 309
fat
effect of cholestyramine on, 292
in jejunal diverticulosis, 299
in pancreatic insufficiency, effect of pancreatin on, 71
in tumor-bearing rats, 53
fluorides, 259
iron
in liver disease, 83
in mink fed raw fish, 143
magnesium, in kwashiorkor, 38
malabsorption syndromes, 138
nutrients, from jejunal pouch prosthesis, after gastrectomy, 300
selenium, effect of calcium on, 330
spectra, of vitamin B_{12} coenzymes, 283
thiamine, effect of penicillin on, 60
tocopherol, effect of alfalfa factor on, 189
vitamin B_{12}
in jejunal diverticulosis, 299
in pyridoxine deficiency, 122
relation to thyroid hormone, 274
species specificity of intrinsic factor on, 177
zinc, effect of calcium on, 111
Acetate
conversion to cholesterol, effect of dietary protein on, 113
Achromotrichia
in mink fed raw fish, 143
Acidity
urine, relation to kidney stones in vitamin B_6 deficiency, 88
Acidosis
bone sodium in, 152
ACTH (*see* **Hormones**)
Activity
physical, relation to growth rate and age, 247
Adenine
in cobamide coenzymes, 85
Adiphenine
derivative, effect on blood cholesterol level, 251
Adipose
tissue, glucose uptake by, effect of chromium on, 348
Adrenal
changes
in normal versus alloxan diabetic rats, on atherogenic diets, 216
in pantothenic acid deficiency, 156
cortex, pantothenic acid and coenzyme A in, effect of ACTH on, 79
function, effect of triparanol on, 262
iron in, in experimental hemochromatosis, 83
removal, effect on vitamin A and ubiquinone in liver, 218
weight
effect of cortisol and pyridoxine deficiency on, 22
effect of dietary cholesterol on, 184
effect of essential fatty acid deficiency on, N127
relation to growth rate and age, 247
Age
differences, in susceptibility to starvation ulcer, 151
relation to fluoride content of teeth, 124
relation to height and weight in U.S. adults, SA193
relation to intestinal flora, in the pig, 47
relation to sodium content of bone, 152
relation to vitamins A and E blood levels, in man, 101
Agriculture
nitrogen fixation by plants, N191
Albumin
binding of vitamin A by, 344
blood levels
in jejunal diverticulosis, 299
in the hippopotamus, N31
copper complex, effect on kidney, 11
metabolism, in protein-losing disorder, 236
Alcohol
consumption, relation to incidence of atherosclerosis, 37
dehydrogenase, in liver, zinc containing, 4
liver cirrhosis from, in the Bantu, SA257
postalcoholic cirrhosis, zinc metabolism in, 4
Alcoholism
cardiomyopathy in, 268
relation to hemochromatosis, 83
Alfalfa
carotene in, 172
fat soluble factor in, effect on tocopherol absorption, 189
Algae
waterbloom, toxicity of, 145
Algeria
heart disease in, N191
Alloxan Diabetes
increased atherogenesis in, 216
Alpha-Ketoglutaric Acid
in muscle, in kwashiorkor, 272
Altitude
polycythemia, lipid deposition in, 276
Amino Acids
absorption, mechanism of, 16
arginine
effect on calcium absorption, 42, 78
requirement for chicks, 49
balance of, for growth, 186
blood levels, in hemorrhagic shock, N158
composition
egg and milk protein, 175
of fat-mobilizing hormone, 39
distribution, in blood, in pyridoxine dependency, 227
excretion in infants and children, 68
glutamic acid, growth needs for, 186
glutamine, excretion in infants and children, 68
glycine, dietary, effect on oxalate excretion, 88
leucine, excess, effect on pregnancy, 342
lysine
effect on calcium absorption, 42, 78
effect on cotton fur condition in mink, 143

Amino Acids—(*Continued*)
excess, effect on pregnancy, 342
lack in rice protein, 175
methionine
absorption mechanism, 16
metabolism, role of cobamide coenzymes in, 85
phenylalanine, diet restriction, in treatment of phenylketonuria, 234, SA353
requirements, for different breeds of hens, SA225
tryptophan
effect on xanthurenic acid excretion, after hydrazides, 8
loading test in pyridoxine dependency, 227
metabolites of, inhibition of thyroxin metabolism by, 174
tyrosine, effect on cotton fur condition in mink, 143
β-Amino Isobutyric Acid
excretion, in infants and children, 68
Ammonia
blood levels, in man, 206
Anemia
cholesterol feeding, in the rabbit, 184
diverticulosis, treatment with vitamin B_{12} and antibiotics, 299
hemolytic, in premature infants given vitamin K substitutes, 75
in infants born prematurely, 195
in mink fed raw fish, 143
myxedema, vitamin B_{12} absorption in, 274
pantothenic acid deficiency, 156
pernicious, vitamin B_{12} clearance from plasma in, 200
vitamin E deficiency, erythrocyte life-span in, 154
Animal
protein, effect on blood cholesterol levels, 70
Antibiotics
effect on dental caries, 105
in foods, effect on intestinal flora, 317
neomycin
intestinal lesions from, N191
malabsorption syndrome from, 72
penicillin, mechanism of thiamine-sparing, 60
tetracycline, treatment of jejunal diverticulosis, 299
Antibody
formation
in B-vitamin deficiencies, 116
in pyridoxine deficient guinea pigs, 315
Antimetabolites
desoxypyridoxine
convulsions from, 8
effect on oxalate synthesis, 88
isonicotinic hydrazide, effect on oxalate excretion, 88
omega-methyl pantothenic acid, effect on guinea pigs, 156
Antioxidants
relation to vitamin E, in chick diets, 52
vitamin E replacements, 217
Aorta
atherosclerosis in, effect of dietary fats on, 25
beta-glucuronidase activity in, in atherosclerosis, 90
changes, in normal versus alloxan diabetic rats, on atherogenic diets, 216
cholesterol in, in experimental atherosclerosis in the chick, 244
lipids in
fatty acid analysis of, 276
host versus homograft tissue, 338
relation to exercise, 55
sudanophilia in, relation to dietary fat and magnesium, 92
surgical coarctation, effect on experimental atherosclerosis, 179
Appendix
ligation, infection after, in dogs fed antibiotics, 317
Appetite
control
by drugs, 202
by hypothalamus, 182
Arteries
arteritis, in rats, effect of restricted food intake on, 305
lipids in
effect of salt intake on, 331
in atherosclerosis associated with anemia, 276
Arteriovenous Difference
of fatty acids, 115
Ascorbic Acid
blood levels, in pantothenic acid deficiency, in guinea pigs, 156
synthesis
effect of drugs on, 48
in the intestine, SA257
use in treatment of heart disease, SA161
Assay
alanine transaminase, in blood, 120
carotene, in alfalfa, 172
intrinsic factor, by guinea pig absorption study, 177
lactic dehydrogenase, in liver, 44
methoxychlor, in milk, N63
tocopherols, using chicks, 146
vitamin B_6, in infant foods, N223
Atherosclerosis
beta-glucuronidase blood levels in, 90
effect of aortic coarctation on, 179
effect of dietary magnesium on, 92
effect of tetraiodothyroformic acid on, 209
effect of thyroxine on, 92
experimental, relation to exercise, 55
fat clearance in, 207
in London busmen, 328
in miniature swine, effect of dietary fats on, 108
in the chick
meal eating versus nibbling, 213
relation to dietary fat, 25, 244
in the dog
effect of linoleic acid, estradiol, and sitosterol on, 338
physiologic and pathologic studies of, 346
incidence, relation to alcohol consumption, 37
lipid deposition, associated with anemia and polycythemia, 276
myocardial and renal infarcts in, effect of salt intake on, 331
myocardial infarction, relation to exercise, 221
plasma and aorta lipids in, fatty acid analysis of, 276
relation to dietary control in diabetes, SA1
relation to fatty acid types consumed, 333
research, in the U.S.S.R., SA161
Availability
arginine, for the chick, 49
niacin, in wheat, 240
tocopherol, effect of alfalfa factor on, 189
zinc
in feeds, 111
in various compounds, 302

Bacteria
Clostridium tetanomorphum, cobamide coenzymes in, 85
E. Coli, intestinal, effect on growth of mice, 12
-free animals, lack of dental caries in, 105
intestinal
effect of antibiotic containing food on, 317
effect of lactose feeding on, 80
effect of penicillin on thiamine synthesis by, 60
relation to diet and age, in the pig, 47
relation to growth of mice, 12
Ballistocardiogram
changes, in canine atherosclerosis, 346
Bantu
disease rarities in, SA257
Barium
Ba^{140}, milk, seasonal variations, 253
Basal Metabolic Rate
in obesity, SA289
Beef
irradiation, effect on vitamin K, 14, 110
Bile Acid
sequestering, by cholestyramine, effect on blood cholesterol level, 292
Bile Salts
need for carotene to vitamin A conversion, 334
Biliary Cirrhosis
treatment with cholestyramine, 292
Bilirubin
blood levels
in norethandrolone poisoning, 2
in premature infants given vitamin K substitutes, 75
Biological Value
tocopherol forms, 146
trans-linoleic acid, as essential fatty acid, 62
Bitot's Spots
relation to vitamin A nutrition, 104, N350
Bladder
stones, in Indians, SA257
Blood
coagulation, in rats fed irradiated beef, 14
erythrocyte counts, in pantothenic acid deficiency, 156
fibrinolytic activity, in cirrhosis, 37
pressure, in canine atherosclerosis, 346
studies, in infants born prematurely, 195
volume, in vitamin B_6 deficiency, 149
Body Composition
measurement by K^{40} content, 134
of infants, of diabetic mothers, 266
relation to meal eating versus nibbling, 9
relation to survival in starvation, 155
Bone
abnormalities, in zinc deficiency, in chicks, 111, 302
calcium in
after feeding magnesium salts, 212
during immobilization, 42
in osteoporosis, 269
density, relation to fluoride in drinking water, 198
development, relation to growth rate and age, 247
fluoride in, 124
magnesium in
changes with dietary magnesium, 178
in kwashiorkor, 38
selenium in, effect of calcium on, 330
sodium in, 152
Sr^{90} to calcium ratio in, 245

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Brain
copper in, after injection, 11
pathology, in canine atherosclerosis, 346
Breast Feeding
relation to incidence of kwashiorkor, 324

Cadmium (*see* **Trace Elements**)
Calcium
absorption
effect of exercise on, 42
effect of lysine on, 78
balance, in man, after treatment with growth hormone from different species, 103
blood levels
in hypercalcemic syndrome, 135
in hypervitaminosis D, N158
in hypogammaglobulinemia, 138
in milk fever, 141
deposition in kidney, after copper injection, 11
dietary, relation to Sr^{90}, 245
effect on fluoride absorption, 259
effect on magnesium requirement, 178
effect on selenium utilization by swine, 330
effect on zinc absorption, 111
in bone, relation to fluoride intake, 124
in milk, seasonal variations, 253, 308
metabolism, in osteoporosis, 269
relation to Sr^{90} in foods, 164
requirement, for different breeds of hens, SA225
Calf
magnesium deficiency in, relation to dietary tocopherol and cod liver oil, 212
Caloric Intake
of London busmen, 328
Caloric Values
from food tables, accuracy of, 6
Cancer
carcinogens in food, discussion, 227
fat metabolism in, 53
from iron-dextran therapy, 195
incidence, relation to water hardness, 132
pancreas, absorption defects in, 138
plasma unesterified fatty acids in, 137
thyroid, incidence, relation to iodine treatment, 166
Cape Town
kwashiorkor studies in, 324
Carbohydrate
absorption
from jejunal pouch prosthesis, after gastrectomy, 300
in non-tropical sprue, 138
component of cobamide coenzymes, 85
dietary
relation to intestinal flora, in the pig, 47
restriction, effect on excretion of fat-mobilizing hormone, 39
glucose
blood levels
changes during work, 23
effect of fat-mobilizing hormone on, 39
in fasting ketosis, 187
in zinc deficiency, 111
hepatic output, effect of insulin on, N286
oxidation, in meal eating versus nibbling animals, 9
use in parenteral fluid therapy, for infantile diarrhea, 296
glucose tolerance
changes from niacin therapy, 325
in cancer, 137
in hypogammaglobulinemia, 138
glucose tolerance factor, 348
glycogen
in liver
effect of cortisol and pyridoxine deficiency on, 22
in glycogen storage disease, N127
in rats force fed with plant proteins, 118
hexoseamine, in metallothionein, 340
lactose, effect on calcium absorption, 42
metabolism, effect of exercise on, 42
pectin, effect on liver and blood cholesterol, 309
starch, effect on pH of cecum, 80
sucrose, use in survival rations, 73
supplementation, effect on work output, 23
xylulose, excretion, after drugs, 48
Carbon Dioxide
rate of production, from different chain length fatty acids, 314
Carbonate
in bone, relation to fluoride intake, 124
Caries (*see* **Teeth**)
Carotene
blood levels, in man, 101
conversion to vitamin A, by the intestine, 334
in alfalfa, 172
Carotenoid
blood level, effect of dietary cod liver oil on, 212
Casein
dietary
relation to arginine requirement for the chick, 49
relation to oxalate excretion, 88
Catecholamines
metabolism, relation to environmental temperature, N255
Cattle
evaluation of vitamin A status in, 172
feeds, use of chlorpromazine on, 77
radiosensitivity to I^{131}, 237
utilization of carotene by, 172
utilization of early and late forage by, 170

Cells
liver, number, in prolonged protein depletion, 285
Cereal Grains
bound niacin in, 240
source of Sr^{90}, 245
vitamin B_6 in, N223
Ceroid
deposition, in children with cystic fibrosis, 168
Ceruloplasmin
synthesis, in Wilson's disease, 133
Cesium
Cs^{137}, sources in diet, 245
Cheese
Sr^{90} content, in England, 164
Chick
arginine requirements for, 49
assay, for tocopherols, 146
beta-glucuronidase blood levels in, in experimental atherosclerosis, 90
breed differences in thiamine content of eggs, SA225
cholesterol synthesis in, effect of dietary protein on, 113
embryos, effect of zinc deficiency on, 148
experimental atherosclerosis in, 25, 244
meal eating versus nibbling, relation to production of atherosclerosis, 213
vitamin E and selenium needs of, 52
zinc deficiency in, from feeding soybean or sesame, 111
zinc requirements for, 302
Children
amino acid excretion in, 68
cystic fibrosis in, 168, SA353
diabetes in, management of, SA353
growth, relation to obesity, 36
hypercalcemic syndrome in, prognosis, 135
kwashiorkor in
growth rate of, 324
muscle composition in, 272
obesity in, consequences in adulthood, 211
renal failure in, management of, SA353
Chloride
relation to drug-induced heart necrosis, 28
Cholesterol
blood levels
effect of adiphenine derivative on, 251
effect of beta-glucuronidase administration on, 90
effect of cholestyramine on, 292
effect of dietary magnesium on, 92
effect of dietary pectin on, 309
effect of diiodothyroacetic acid on, 250
effect of egg yolk feeding on, 293
effect of ethyl linoleate feeding on, 117
effect of exercise on, 55
effect of meal eating versus nibbling in chicks, 213
effect of niacin on, 325
effect of propylthiouracil on, in thyroidectomized dogs, 179
effect of tetraiodothyroformic acid on, 209
effect of triparanol on, 262
effect of type of dietary protein on, 70
in canine atherosclerosis, 346
in hypogammaglobulinemia, 138
in normal versus alloxan diabetic rats, on atherogenic diets, 216
in swine, after feeding various fats, 108
of London busmen, 328
relation to dietary fat, 25, 184, 244
spontaneous variations in, 100
dietary
effect on organ weights, 184
relation to rate of fatty acid synthesis, 113
esters, blood and aorta levels, relation to exercise, 55
feeding, effect on blood level, 293
in aorta
normal and atherosclerotic, 276
relation to exercise, 55
in liver
effect of dietary fats on, 184
effect of fat-mobilizing hormone on, 39
in thiamine deficiency, 21
labeled, in vitro incorporation into lipoproteins, N95
synthesis
effect of adiphenine derivative on, 251
effect of dietary protein on, 113
effect of triparanol on, 262
in swine, effect of dietary fats on, 108
Cholestyramine
effect on blood cholesterol level, 292
Cholic Acid
dietary, use in production of atherosclerosis, 92
Choline
deficiency
hemosiderin deposits in liver and kidney in, N160
plus iron feeding, to produce experimental hemochromatosis, 83
Chromium
constituent of glucose tolerance factor, 348
Citral
effect on intraocular pressure, N96
Citrate
in bone, relation to fluoride intake, 124
in urine, in vitamin B_6 deficiency, effect of magnesium on, 306
Cobalt (*see* **Trace Elements**)
Cobamide
vitamin B_{12} forms in liver, 283
Cod Liver Oil
dietary, effect on magnesium utilization by the calf, 212
Coenzymes
B_{12} coenzyme, chemistry of, 85, 283
coenzyme A, in adrenal cortex, effect of ACTH on, 79
coenzyme Q, effect on muscular dystrophy in guinea pigs, 20
pyridoxal phosphate
effect on transaminase activity of blood, 120
in liver, effect of riboflavin on, 336
TPNH, role in fat synthesis, 9
Coffee
consumption, in the U. S., SA33
Cold
effect on blood cholesterol levels, 100
effect on coronary arteries and testes, N95
Congenital Malformation
incidence, relation to water hardness, 132
Convulsions
from hydrazides and desoxypyridoxine, 8
in pyridoxine dependency state, in an infant, 227
Copper (*see* **Trace Elements**)
Coprophagy
relation to thiamine-sparing by penicillin, 60
role in vitamin K nutrition, 110

Corn
oil, versus animal fat, in production of atherosclerosis in the chick, 244
protein, force feeding of, kwashiorkor-like results from, 118
Cottonseed Oil
effect on blood fat levels and atherosclerosis, 25
Cow
effects of excess fluorine on, 139
milk, composition of, 308
milk fever in, effect of vitamin D on, 141
milk production by, effect of chlorpromazine on, 77
radiosensitivity to I^{131}, 237
Creatine
dietary, effect on arginine requirement for chicks, 49
Creatinine
clearance
in hypercalcemic syndrome, 135
in potassium deficiency, 242
excretion, in infants and children, 68
Cytochemistry
liver, cell types in, 44

Death Rate
from infectious diseases, SA65
Dehydration
factor in management of heart disease, 35
Dentin
teeth, fluorine in, 139
Diabetes
atherogenesis in, 216
blood saccharoid levels in, N254
dietary control, relation to vascular disease, SA1
from niacin treatment of hypercholesterolemia, 325
in children, management of, SA353
in mother, relation to body composition of offspring, 266
obesity in, control by drugs, 202
phenylketonuria in, 264
Diarrhea
in infants, parenteral fluid therapy for, 296
Diet
analysis, use in evaluating nutritional status, SA321
British, Sr^{90} in, 164
fat-free, effect on blood and liver cholesterol, 184
formulas, for weight reduction, SA289
habits, relation to production of atherosclerosis, 213
London busmen, atherosclerosis in, 328
management, of heart disease, 35
radionuclides in, 245
relation to amino acid excretion in infants, 68
restricted, effect on health and longevity, 305
surveys, methodology, 6
treatment
of heart disease, SA161
of phenylketonuria, 234, SA353
Digestibility
early versus late forages, 170
3,5 Diiodothyroacetic Acid
effect on blood and liver cholesterol, 250
Dinitrophenol
effect on methionine absorption, 16
Disease
angina, vitamin E therapy for, N63
cardiovascular
fat clearance in, 207
incidence, relation to hardness of drinking water, 132
chronic myelocytic leukemia, vitamin B_{12} clearance from plasma in, 200
cystic fibrosis, ceroid deposition in, 168
diverticulosis
absorption defect in, 138
anemia and steatorrhea of, treatment with vitamin B_{12} and antibiotics, 299
fibrocystic, in children, SA353
glycogen storage, liver enzymes in, N127
hemochromatosis, experimental, 83
incidence
in obesity, SA1
relation to food intake, 305
infantile hypercalcemic syndrome, 135
infectious, death rates from, SA65
malabsorption syndromes, 138
Mediterranean fever, fat tolerance in, N352
nephritis, vitamin E therapy for, N63
non-tropical sprue, absorption defects in, 138
osteoporosis, calcium metabolism in, 269
oxaluria, in vitamin B_6 deficiency, 88
phenylpyruvic oligophrenia, diet treatment of, 234
pre-eclampsia, vitamin E treatment of, N63
protein-losing enteropathy, intestinal permeability in, 236
pyridoxine dependency, 227
rheumatic fever, incidence in Algeria, N191
sprue, relation to lesions from neomycin, N191
Whipple's disease, electron microscope studies of macrophages in, 230
white muscle disease, selenium treatment of, 169
Wilson's
copper depletion treatment of, 133
role of copper in, 11
xanthoma tuberosum, treatment with niacin, 325
Dog
aortic coarctation, effect on experimental atherosclerosis, 179
atherosclerosis, physiologic and pathologic studies of, 346
DPPD
vitamin E replacement, 217
Drugs (*see also* **Antibiotics, Antimetabolites**)
adiphenine derivative, effect on blood cholesterol level, 251
amphetamine, for control of obesity, 202
chlorpromazine, effect on milk production in cows, 77
cholestyramine, effect on blood cholesterol level, 292
diiodothyroacetic acid, effect on liver and blood cholesterol, 250
dinitrophenol, effect on methionine absorption, 16
effect on ascorbic acid synthesis, 48
epinephrine, effect on blood cholesterol levels, 100
for weight reduction, discussion, SA289
hydrazides, effect on xanthurenic acid excretion, 8
induction of heart necrosis by, effect of chlorides on, 28
isonicotinic hydrazide, effect on oxalate excretion, 88

penicillamine
effect on amino acid absorption, 16
use in treatment of Wilson's disease, 133
phthalylsulfathiazole, effect on oxalate excretion, 88
procaine, injection in hypothalamus, effect on feeding behavior, 182
propylthiouracil, effect on blood cholesterol in thyroidectomized dogs, 179
tetraiodothyroformic acid, use in hypothyroid patients with hypercholesterolemia, 209
triparanol, effect on blood cholesterol levels, 262
vitamin E, use in nephritis, heart disease, abortion, N63
vitamin K substitutes, toxicity for premature infants, 75

Eating
control, by hypothalamus, 182
habits
of obese people, SA289
relation to production of atherosclerosis, 213
physiology of, research in the U.S.S.R., SA129
Eczema
association with phenylketonuria, 264
Edema
in malnourished infants, 232
use in evaluating nutritional status, SA321
Efficiency
physical, effect of food and water restriction on, 23
Egg
consumption, in the U. S., SA33
diet supplementation of rice with, 175
production, relation to zinc in poultry feed, 302
Sr^{90} content, in England, 164
thiamine in, of different breeds of hens, SA225
vitamin B_6 content, N223
yolk, feeding, effect on blood lipid levels, 293
Electrocardiogram
changes
in alcoholic cardiomyopathy, 268
in canine atherosclerosis, 346
Electroencephalogram
abnormal, in pyridoxine dependent infant, 227
changes, in phenylketonuria, 234
Electrolytes
in muscle, in kwashiorkor, 272
use in parenteral fluid therapy, for infantile diarrhea, 296
Electron microscope
studies of intestine, in Whipple's disease, 230
Embryo
chick, malformations in, in zinc deficiency, 148
Enamel
teeth, fluorine in, 139
Energy
metabolism, history of research in Soviet Union, SA161
England
diet in, Sr^{90} content, 164
Environment
low temperature, effect on coronary arteries and testes, N95
relation to nutritive values of food, research in the U.S.S.R., SA129
Enzymes
alcohol dehydrogenase, in liver, zinc containing, 4
alkaline phosphatase
blood level, effect of adiphenine derivative on, 251
blood levels, in norethandrolone poisoning, 2
in intestine, in zinc deficiency, 111
beta-glucuronidase, administration, in atherosclerosis, 90
cytochrome oxidase, presence of copper in, 80
fructose-1,6 diphosphatase, in liver, in glycogen storage disease, N127
glucose-6-phosphatase, in liver, in glycogen storage disease, N127
glutamate isomerase, role of vitamin B_{12} coenzyme in, 283
hexosemonophosphate dehydrogenase, in meal eating versus nibbling animals, 9
lactic dehydrogenase
in liver
effect of starvation on, 44
in glycogen storage disease, N127
lipoprotein lipase, in tumor-bearing rats, 53
lipoxidase, effect on mucopolysaccharide synthesis in colon tissue, 91
pancreatin, use in test for malabsorption syndromes, 71
phosphoglucomutase, in liver, in glycogen storage disease, N127
phosphohexoseisomerase, in liver, in glycogen storage disease, N127
pyridoxal phosphate oxidase, in liver, effect of riboflavin feeding on, 336
succinic dehydrogenase, tissue levels in iron deficiency, 27
transaminase
blood levels
in norethandrolone poisoning, 2
in pyridoxine deficiency, 120
in pyridoxine dependency, 227
in vitamin B_{12} deficiency, 122
in white muscle disease, 169
in liver, effect of cortisol and pyridoxine deficiency on, 22
tryptophan pyrrolase, inhibition by serotonin, 174
Epinephrine (*see* **Hormones**)
Erythrocyte
life-span, in vitamin E deficient monkeys, 154
Ethoxyquin
vitamin E replacement, 217
Eye
Bitot's spots, relation to vitamin A nutrition, 104
color, in phenylketonuria, 264
copper deposition in, in Wilson's disease, effect of penicillamine on, 133
development, relation to growth rate and age, 247
examination, use in evaluating nutritional status, SA321
glaucoma, production by citral, N96
Exercise
effect on blood ammonia levels, 206
effect on calcium absorption, 42
relation to development of experimental atherosclerosis, 55
relation to myocardial infarction, in atherosclerosis, 221
Extracellular Volume
in infants of diabetic mothers, 266

FAO
program for food relief, SA33

Fasting
effect on excretion of fat-mobilizing hormone, 39
ketosis, in pregnant guinea pigs, 187
Fat
absorption
from jejunal pouch prosthesis, after gastrectomy, 300
in jejunal diverticulosis, 299
in malabsorption syndromes, 138
in non-tropical sprue, 138
in pancreatic insufficiency, effect of pancreatin on, 71
animal, versus corn oil, in production of atherosclerosis in the chick, 244
balance, after cholestyramine, 292
blood levels
after beta-glucuronidase administration, 90
after cottonseed oil feeding, 25
after egg yolk feeding, 293
after ethyl linoleate feeding, 117
in meal eating versus nibbling chicks, 213
in normal versus alloxan diabetic rats, on atherogenic diets, 216
in swine, after feeding various fats, 108
regulation by hypothalamus, N222
relation to exercise, 55
spontaneous variations in, 100
body
composition, relation to diet, 275
in infants of diabetic mothers, 266
measure of obesity, SA289
measurement by K^{40} content, 134
relation to growth rate and age, 247
relation to survival in starvation, 155
deficiency, effect on pituitary and testes, 17
deposition
in aorta, effect of fatty acids, estradiol and sitosterol on, 338
in arteries, effect of salt intake on, 331
dietary
effect on fluoride retention, 259
effect on vitamin B_6 metabolism, 336
relation of fatty acids to essential fatty acid deficiency, N127
relation to atherosclerosis in the chick, 25
relation to blood and liver cholesterol levels, 184
I^{131} fat-pancreatin test, for malabsorption syndromes, 71
intake, of London busmen, 328
intravenous, clearance from blood, 207
liver
effect of dietary pectin on, 309
effect of diiodothyroacetic acid on, 250
effect of fat-mobilizing hormone on, 39
in rats force fed with plant proteins, 118
in thiamine deficiency, 21
metabolism
abnormal, in obesity, SA289
in tumor-bearing rats, 53
mobilizing hormone, nature and properties, 39
skin, measure of obesity in children, 36
stores, in pantothenic acid deficiency, 156
tolerance test, in tumor-bearing rats, 53
tolerance to, in Mediterranean fever, N352
types, in normal and atherosclerotic aorta, 276
vegetable, effect on atherosclerosis in chicks, 25
Fatty Acids
dietary, relation of chain length to depot fat, 275
distribution, in milk, seasonal variations in, 308
essential
deficiency disease, relation to type of dietary fat, N127
role in cholesterol synthesis, 184
esters, blood levels, effect of type of dietary protein on, 70
ethyl linoleate, effect on blood and carcass cholesterol levels, 117
in plasma and aorta lipids, 276
linoleic acid
atherogenic effects of, 333
effect on depot fat, 275
oxidation rates, effect of chain length on, 314
synthesis, effect of dietary protein on, 113
trans-linoleic, essential fatty acid activity of, 62
unesterified, plasma levels in cancer, 137
unsaturated, effect on lipid deposition in aorta, 338
uptake, by skeletal muscle, 115
Feather
abnormalities, in poultry with zinc deficiency, 302
Feces
thiamine in, after penicillin feeding, 60
tocopherol in, effect of alfalfa factor on, 189
Feeds
alfalfa, carotene content of, 172
cattle, use of chlorpromazine in, 77
early versus late forage, utilization, 170
efficiency, in the pig, effect of lactose diet on, 47
poultry, zinc needs in, 302
swine, sesame and soybean meals, zinc deficiency from, 111
Fertility
in cows, after I^{131} damage, 237
maintenance by antioxidants in absence of vitamin E, 217
mink, effect of whale liver diet on, N318
Fetus
growth, in copper deficiency, 28
Fish
raw, effect of feeding on iron absorption, 143
Flavin Mononucleotide
effect on pyridoxal phosphate level in the liver, 336
Flour
Sr^{90} content, in England, 164
Fluoride (*see* **Trace Elements**)
Folacin
absorption, in non-tropical sprue, 138
requirement, N159
Food
additives, discussion, SA97, 227, N351
analysis, use in diet surveys, 6
consumption
effect of hypothalamic stimulation on, 182
in experimental hypertension, 57
in meal eating versus nibbling chicks, 213
in the U. S., SA33
restricted
effect on health and longevity, 305
effect on work output, 23
use in evaluating nutritional status, SA321
faddism, SA1
fluorides in, 259
habits
in childhood and adulthood, 211
of London busmen, 328
relation to body composition, 9
relation to kwashiorkor, 324
relation to production of atherosclerosis, 213
pemmican, use as emergency ration, 73

requirements, for space flight, activities of the Food and Nutrition Board, SA97
research, in the U.S.S.R., SA129
sources of radionuclides, 164, 245, 253
Food and Nutrition Board
functions of, SA97
Food Values
nutrients, in milk, 306
tables, use in diet surveys, 6
vitamin B_6, in milk, N223
Forage
early versus late, utilization, 170
Force Feeding
effect on body composition, 9
plant proteins, kwashiorkor-like results from, 118
value after injury, 123
Formic Acid
metabolism, role of cobamide coenzymes in, 85
Fruit
vitamin B_6 content, N223

Genetics
influence on nutrient requirements of hens, SA225
influence on tooth characteristics, 311
relation to nutritive values of food, research in the U.S.S.R., SA129
Geriatrics
bone fracture in the aged, relation to fluoride intake, 198
Globulin
binding of vitamin A by, 344
blood levels, in the hippopotamus, N31
gamma, blood levels, in vitamin B_6 deficiency, 149
hypogammaglobulinemia, absorption defects in, 138
Glucose (*see* Carbohydrate)
Glucose tolerance factor, 348
Glucuronic acid
blood saccharoid substance, N254
synthesis, effect of drugs on, 48
Glycogen (*see* Carbohydrate)
Glycoprotein
accumulation, in Whipple's disease, 230
Goiter
research in the U.S.S.R., SA129
treatment, relation to incidence of thyroid cancer, 166
Growth
children
in hypercalcemic syndrome, 135
relation to obesity, 36
with kwashiorkor, 324
cirrhotic liver, after partial hepatectomy, N319
decrease, after injury, 123
hormone (*see* **Hormones**)
mice, effect of intestinal parasites on, 12
premature infants, effect of norethandrolone on, 2
relation to body weight and functions, 247
response, from glutamic acid, 186
Guinea Pig
intestine, use in assay of intrinsic factor, 177
pantothenic acid deficient, blood studies in, 156
pregnant, fasting ketosis in, 187
pyridoxine deficient, antibody production in, 315
vitamin E deficiency in, 20
Gulonic Acid
synthesis, effect of drugs on, 48

Gynecomastia
in the Bantu, SA257
Grass
protein, for human food, N31

Hair
changes, after triparanol treatment, 262
color, in phenylketonuria, 264
Hatchability
chicken eggs, effect of zinc deficiency on, 148
Heart
calcium in, after feeding magnesium salts, 212
changes, in normal versus alloxan diabetic rats, on atherogenic diets, 216
cholesterol in, after feeding various fats, 108
coronary arteries, effect of lowered environmental temperature on, N95
disease
dietary management, 35
in Algeria, N191
incidence, relation to hardness of drinking water, 132
vitamin E therapy for, N63
examination, use in evaluating nutritional status, SA321
iron in, in experimental hemochromatosis, 83
lesions, in Whipple's disease, 230
lipids, in atherosclerosis associated with anemia, 276
myocardial degeneration, in rats, effect of restricted food intake on, 305
myocardial fibrosis, incidence in low temperature environment, N95
myocardial infarction
effect of salt intake on, 331
fat clearance after, 207
in the Bantu, SA257
relation to dietary fat fed, 333
relation to exercise in atherosclerosis, 221
relation to portal cirrhosis, 37
myopathy, in alcoholics, 268
necrosis, effect of chlorides on, 28
pathology, in canine atherosclerosis, 346
selenium in, effect of calcium on, 330
succinic dehydrogenase in, in iron deficiency, 27
ubiquinone in, in vitamin A deficiency, 218
weight
in essential fatty acid deficiency, N127
in iron deficiency, 27
Height-Weight
data
for estimation of obesity, SA289
U. S. adults, SA193
examinations, use in evaluating nutritional status, SA321
Hemochromatosis
experimental production of, 83
Hemoglobin
levels
in infants born prematurely, 195
in iron deficiency, 27
in pantothenic acid deficiency, 156
in vitamin B_6 deficiency, 149
in zinc deficient poultry, 302
Hemorrhage
from feeding irradiated beef, 14, 110
Hemosiderin
in liver, in experimental hemochromatosis, 83
in liver and kidney, in choline deficiency, N160

Heredity
breed differences in thiamine content of eggs, SA225
relation to tooth decay, 105
Hippopotamus
serum protein levels in, N31
Histology
adrenal cortex, changes after ACTH, 79
liver, cell types, 44
Hormones
ACTH, effect on pantothenic acid and coenzyme A in adrenal cortex, 79
adrenal, synthesis, effect of triparanol on, 262
androgen, lack in fat deficiency, 17
cortisol, effect on liver transaminase, 22
epinephrine, inhibition of tryptophan pyrrolase by, 174
estradiol, effect on lipid deposition in aorta, 338
fat-mobilizing
in cancer, 137
nature and properties, 39
gonadotrophin, effect on testicular changes in fat deficiency, 17
growth, from various species, activity in man, 103
insulin
effect of chromium on, 348
effect on hepatic output, N286
sensitivity, in cancer, 137
noradrenaline, metabolism, relation to environmental temperature, N255
norethandrolone, effect on growth of premature infants, 2
pituitary extract, effect on testicular changes in fat deficiency, 17
pituitrin, to produce myocardial infarction in atherosclerosis, 221
steroid, effect on location of starvation ulcers, 151
testosterone, effect on testicular changes in fat deficiency, 17
thyroxine, conversion to triiodothyronine, inhibition by serotonin, 174
thyroxine, effect on experimental atherosclerosis, 92
Horse
kidney, cadmium in, 340
Hydrocarbons
effect on ascorbic acid synthesis, 48
5-Hydroxytryptophan
inhibition of thyroxine metabolism by, 174
Hyperphagia
control, by drugs, 202
Hypertension
coronary, effect on experimental atherosclerosis, 179
dietary management, 35
experimental, aversion to salts in, 57
incidence, relation to water hardness, 132
Hypervitaminosis A
from eating whale liver, N318
Hypervitaminosis D
blood studies in, N158
Hypogammaglobulinemia
absorption defects in, 138
Hypothalamus
blood fat regulating center in, N222
stimulation, effect on feeding behavior, 182
Hypothyroidism
vitamin B_{12} absorption in, 274
India
leaf protein as food, N31
occurrence of bladder stones in, SA257
Infants
amino acid excretion in, 68
cystic fibrosis in, ceroid deposition in, 168
diarrhea in, parenteral fluid therapy for, 296
diet, Sr^{90} content, in England, 164
foods for, vitamin B_6 in, N223
hypercalcemic syndrome in, prognosis, 135
kwashiorkor in, magnesium metabolism in, 38
malnourished, body water in, 232
newborn, temperature regulation in, relation to noradrenaline metabolism, N255
nutrition, studies by Food and Nutrition Board, SA97
of diabetic mothers, body composition of, 266
phenylketonuria, diet treatment of, 234
premature
effect of norethandrolone on growth, 2
iron deficiency in, 195
toxicity of vitamin K substitutes for, 75
pyridoxine dependency in, 227
Infection
after appendix ligation, in dogs fed antibiotics, 317
relation to tooth decay, 105
role in kwashiorkor, 324
Injury
growth decrease and nitrogen deficits after, 123
Insecticides
methoxychlor, residue in milk, N63
Insulin (*see* **Hormones**)
Intelligence Quotient
in children with phenylketonuria, 234
Intestine
absorption of amino acids, mechanism of, 16
absorption of calcium from different segments, 78
bacteria
effect of antibiotic containing foods on, 317
effect of lactose feeding on, 80
effect of penicillin on thiamine synthesis by, 60
in the pig, relation to diet and age, 47
bile acid sequestering in, effect on blood cholesterol level, 292
carotene to vitamin A conversion in, 334
changes
from neomycin, 72, N191
in Whipple's disease, 230
colon, mucopolysaccharide synthesis in, in vitamin A deficiency, 91
diverticulosis, absorption defects in, 138
guinea pig, use in assay of intrinsic factor, 177
iron in, in experimental hemochromatosis, 83
jejunal diverticulosis, anemia and steatorrhea of, treatment with vitamin B_{12} and antibiotics, 299
parasites in, effect on growth of mice, 12
permeability, in protein-losing enteropathy, 236
selenium in, effect of calcium on, 330
weight, relation to growth rate and age, 247
Intrinsic Factor
in stomach, in myxedema, 274
species specificity, for vitamin B_{12} absorption, 177
Inulin
clearance, in potassium deficiency, 242
Iodine (*see* **Trace Elements**)
Iron
absorption
in liver disease, 83

in mink fed raw fish, 143
in non-tropical sprue, 138
deficiency
effect on tissue succinic dehydrogenase levels, 27
in prematurely born infants, 195
feeding, to produce experimental hemochromatosis, 83
in liver, in hemochromatosis, 83
Iron-Dextran
treatment of infants with anemia, 195
Irradiation
beef, effect on vitamin K, 14, 110
Isocitrate
in muscle, in kwashiorkor, 272

Ketone
blood levels
effect of fat-mobilizing hormone on, 39
in hemorrhagic shock, N158
in pregnancy, 187
Kidney
changes
in normal versus alloxan diabetic rats, on atherogenic diets, 216
in pantothenic acid deficiency, 156
failure, in children, management of, SA353
function
in hypercalcemic syndrome, 135
in potassium deficiency, 242
glomerulonephritis, incidence in low temperature environment, N95
hemosiderin in, in choline deficiency, N160
infarcts, effect of salt intake on, 331
metallothionein, cadmium and zinc in, 340
nephritis
in rats, effect of restricted food intake on, 305
vitamin E therapy for, N63
oxalate stones, relation to magnesium and vitamin B_6 intake, 306
pathology
in canine atherosclerosis, 346
in copper poisoning, 11
selenium in, effect of calcium on, 330
stones, in vitamin B_6 deficiency, 88
succinic dehydrogenase in, in iron deficiency, 27
vitamin B_{12} in, relation to thyroid activity, 274
weight
in essential fatty acid deficiency, N127
in rats force fed with plant proteins, 118
relation to growth rate and age, 247
Kwashiorkor
follow-up studies, in Cape Town, 324
from force feeding of plant proteins, 118
magnesium metabolism in, 38
muscle composition in, 272
Kynurenine
formation, from tryptophan, inhibition by serotonin, 174

Lactalbumin
diet supplementation of rice with, 175
Lactate
blood levels, in zinc deficiency, 111
in muscle, in kwashiorkor, 272
use in parenteral fluid therapy, in infantile diarrhea, 296
Lactation
dietary protein lack during, effect on teeth of offspring, 311
effect on milk fever, 141
in cows, after I^{131} damage, 237
in human milk, variations in, 308
Lactose (*see* **Carbohydrate**)
Leaf
proteins, for human food, N31
Learning
ability, relation to growth rate and age, 247
Letters to the Editor
Guha, B. C., 31
Oomen, H. A. P. C., 350
Sheehy, T. W., 191
Shute, E. V., 63
Life Span
at low temperatures, N95
erythrocytes, in vitamin E deficient monkeys, 154
recent changes in, SA65
relation to food intake, 305
Linseed oil
effect on blood fat levels and atherosclerosis, 25
Lipoprotein
binding of vitamin A by, 344
blood levels
effect of niacin on, 325
in normal versus alloxan diabetic rats, on atherogenic diets, 216
of London busmen, 328
spontaneous variations in, 100
in vitro incorporation of labeled cholesterol in, N95
Liver
beef, vitamin B_6 content, N223
carotenoids, correlation with blood vitamin A, 172
cell types in, 44
ceroid deposition in, in children with cystic fibrosis, 168
changes
in normal versus alloxan diabetic rats, on atherogenic diets, 216
in pantothenic acid deficiency, 156
cholesterol in
after feeding ethyl linoleate, 117
effect of dietary pectin on, 309
effect of diiodothyroacetic acid on, 250
in swine, after feeding various fats, 108
on fat-free diet, 184
cholesterol synthesis in, effect of dietary protein on, 113
cirrhosis
alcoholic, in the Bantu, SA257
blood ammonia levels in, 206
postalcoholic, zinc metabolism, 4
regenerative ability, N319
relation to iron absorption, 83
copper in, effect on kidney, 11
damage, from norethandrolone, 2
disease, blood ammonia levels in, 206
enzyme levels, in glycogen storage disease, N127
fat
effect of dietary pectin on, 309
effect of fat-mobilizing hormone on, 39
effect of thiamine deficiency on, 21
fat synthesis
in meal eating versus nibbling animals, 9
fatty, from feeding adiphenine derivative, 251
function
after niacin treatment of hypercholesterolemia, 325
effect of adiphenine derivative on, 251
effect of triparanol on, 262

Liver—(*Continued*)
in Wilson's disease, effect of penicillamine on, 133
glucose output by, effect of insulin on, N286
glycogen, effect of cortisol and pyridoxine deficiency on, 22
hemosiderin in, in choline deficiency, N160
hepatolenticular degeneration
role of copper in, 11
treatment of copper depletion, 133
iron in
after choline deficiency and iron feeding, 83
in hemochromatosis, 83
lactic dehydrogenase in, effect of starvation on, 44
lesions, in Whipple's disease, 230
nitrogen, in prolonged protein depletion, 285
pathology, in canine atherosclerosis, 346
peliosis hepatitis, from norethandrolone, 2
portal cirrhosis, relation to myocardial infarction, 37
protein, binding of vitamin A by, 344
pyridoxal phosphate in, effect of riboflavin feeding on, 336
selenium in, effect of calcium on, 330
succinic dehydrogenase in, in iron deficiency, 27
tocopherols in, in the chick, assay procedure, 146
transaminase in, effect of cortisol and pyridoxine deficiency on, 22
ubiquinone in, in vitamin A deficiency, 218
vitamin B_{12} coenzyme in, 283
vitamin B_{12} in, relation to thyroid activity, 274
weight
changes in starvation, 44
effect of cortisol and pyridoxine deficiency on, 22
effect of dietary cholesterol on, 184
in essential fatty acid deficiency, N127
in iron deficiency, 27
in prolonged protein depletion, 285
in rats force fed with plant proteins, 118
in thiamine deficiency, 21
relation to growth rate and age, 247
whale, toxicity, N318
London
busmen, atherosclerosis in, 328
Longevity
relation to food intake, 305
Lung
changes, in normal versus alloxan diabetic rats, on atherogenic diets, 216
iron in, in experimental hemochromatosis, 83
selenium in, effect of calcium on, 330
Lymph Nodes
lesions, in Whipple's disease, 230

Magnesium
blood levels
effect of dietary fats on, 92
in normal versus alloxan diabetic rats, on atherogenic diets, 216
deficiency
in muscular dystrophy in sheep, 41
in the calf, relation to dietary tocopherol and cod liver oil, 212
effect on experimental atherosclerosis, 92
effect on fluoride absorption, 259
effect on oxalate deposition in vitamin B_6 deficiency, 88
in bone, relation to fluoride intake, 124
in muscle, in kwashiorkor, 272
intake, relation to oxalate kidney stones, 306
metabolism, in protein malnutrition, 38
requirement, for the rat, 178
Maize
bound niacin compounds in, 240
Malabsorption Syndrome
diagnosis, by I^{131} fat-pancreatin test, 71
discussion, 138
from neomycin, 72
vitamin B_{12} clearance from plasma in, 200
Malformation
chick embryos, in zinc deficiency, 148
Malnutrition
in infants, body water in, 232
malignant, muscle composition in, 272
physical signs of, SA321
protein
magnesium metabolism in, 38
studies by Food and Nutrition Board, SA97
relation to hemochromatosis, 83
Manganese (*see* **Trace Elements**)
Marasma
body water in, 232
Mastication
investigations of, in the U.S.S.R., SA129
Meat
consumption, in the U.S., SA33
source of Cs^{137}, 245
Mediterranean Fever
fat tolerance in, N352
Melanin
formation, in phenylketonuria, 264
Metabolic Rate (*see* **Basal Metabolic Rate**)
Metallothionein
cadmium and zinc in, 340
Methoxychlor
residue in milk, N63
β-Methylaspartic Acid
role in animal metabolism, 85
Milk
diet supplementation of rice with, 175
fat, consumption, in the U.S., SA33
human and cow, composition, 306
in diet, effect on amino acid excretion in infants, 68
methoxychlor residue in, N63
production
in cows
after I^{131} damage, 237
effect of chlorpromazine on, 77
relation to early versus late forage, 170
source of radionuclides, 164, 245, 253
standards, Food and Nutrition Board, SA97
vitamin B_6 in, N223
Milk Fever
effect of vitamin D on, 141
Milo
protein, force feeding of, kwashiorkor-like results from, 118
Mink
iron absorption in, effect of raw fish diet on, 143
toxicity of whale liver for, N318
Monkey
eating habits, relation to production of atherosclerosis, 213
growth hormone from, activity in man, 103
vitamin E deficiency in, erythrocyte life-span in, 154
Mortality Rate
in phenylketonuria, 264
Mucopolysaccharide
synthesis, in colon tissue, in vitamin A deficiency, 91

Muscle
calcium in, after feeding magnesium salts, 212
ceroid deposition in, in children with cystic fibrosis, 168
composition, in kwashiorkor, 272
dystrophy
in guinea pigs, 20
in sheep, origins of, 41
fatty acid uptake by, 115
heart, importance of chlorides for, 28
magnesium in, in protein malnutrition, 38
muscular dystrophy, in rats, effect of restricted food intake on, 305
release of ammonia from, in exercise, 206
Sr^{90} to calcium ratio in, 245
weight, in rats force fed with plant proteins, 118

Nerve
function, in Wilson's disease, effect of penicillamine on, 133
system, role in atherosclerosis, SA161

Niacin
bound, in wheat, 240
effect on blood cholesterol levels, 325

Nibbling
versus meal eating, effect on body composition, 9

Nitrogen
balance
after injury, 123
in malnourished infants, 232
in man, after treatment with growth hormone from different species, 103
excretion, in infants and children, 68
fixation, in plants, N191
in liver
effect of starvation on, 44
prolonged protein depletion, 285
metabolism, effect of exercise on, 42

Notes
A Hypothalamic Fat Center, 222
American Board of Nutrition Announcement, 31
Announcement, 288
Blood Saccharoids, 254
Blood Serum Proteins of the Hippopotamus, 31
Catalog of Films and Filmstrips on Food and Nutrition, 287
Chemicals in Food, 351
Citral, Vitamin A, and Glaucoma, 96
Dietary Treatment of Familial Mediterranean Fever, 352
Environmental Temperatures and Urinary Metabolites of Noradrenaline, 255
Fatty Acid Chain Length and Essential Acid Deficiency, 127
Heart Disease in Algeria, 191
Hypervitaminosis D, 159
Life Span and Pathology of Rats Maintained at 6°C and 30°C, 95
Liver Enzymes in Glycogen Storage Disease, 127
Liver Regeneration After Partial Hepatectomy in Cirrhotic Rats, 319
Methoxychlor—Not A Milk Residue Problem, 63
Nitrogen Fixation by Nonleguminous Plants, 191
Radioactive Cholesterol Labelling of Serum Lipoproteins, 95
The Daily Dose of Folacin, 159
The Effect of Insulin on Hepatic Glucose Output in Man, 286
Vitamin A Toxicity in Whale Liver, 318
Vitamin B_6 Content of Infant Foods, 223

Nutrition
education
activities of the Food and Nutrition Board, SA97
for obesity and diabetes, SA1
relation to maturation in children, 36
research, in the U.S.S.R., SA129, SA161
status, methods of evaluating, SA321
survey
methodology, 6
of body water in malnourished infants, 232
of calcium intake, 269
of London busmen, 328
of rheumatic fever in Algeria, N191
of vitamin A nutrition and Bitot's spots, 104

Obesity
control by drugs, 202
in childhood and adulthood, 211
in children, relation to growth and maturation, 36
in infants of diabetic mothers, 266
measurement by body K^{40} content, 134
measurements of, SA289
weight reduction programs for, SA1

Obstructive Jaundice
treatment with cholestyramine, 292

Oil
vegetable, effect on atherosclerosis in chicks, 25

Osteoporosis
incidence, relation to fluoride intake, 198

Oxalacetate
in muscle, in kwashiorkor, 272

Oxalate
excretion, in vitamin B_6 deficiency, 88
kidney stones, relation to magnesium and vitamin B_6 intake, 306

Oxaluria
in vitamin B_6 deficiency, 88

Oxidation
rates, of different chain length fatty acids, 314

Pancreas
cancer, absorption defect in, 138
cystic fibrosis, ceroid deposition in, 168
insufficiency, fat absorption in, effect of pancreatin on, 71
iron in, in experimental hemochromatosis, 83

Pantothenic Acid
deficiency
antibody formation in, 116
in guinea pigs, blood studies in, 156
in adrenal cortex, effect of ACTH on, 79

Parathyroid
function, in milk fever, 141

Parakeratosis
in swine, 111

Pathology
alcoholic cardiomyopathy, 268
canine atherosclerosis, 346
experimental hemochromatosis, 83
heart, from drugs, effect of chlorides on, 28
hypercalcemic syndrome, 135
intestinal mucosa, after neomycin, 72
intestine
from neomycin, N191
in protein-losing disorder, 236
in Whipple's disease, 230
kidney
in copper poisoning, 11
in potassium deficiency, 242

Pathology—(*Continued*)
liver
from norethandrolone, 2
in rats force fed with plant proteins, 118
low temperature environment, N95
magnesium deficiency, in the rat, 178
muscle, in muscular dystrophy of sheep, 41
pantothenic acid deficiency, in guinea pigs, 156
pituitary, in fat deficiency, 17
pyridoxine dependency, in an infant, 227
testes, in fat deficiency, 17
vitamin K substitute poisoning, in premature infants, 75
waterbloom poisoning, 145
Pectin
dietary, effect on liver and blood cholesterol, 309
Pemmican
as emergency ration, 73
Penicillin (*see* **Antibiotics**)
Peptide
nature, fat-mobilizing hormone, 39
Permeability
of intestine, in protein-losing enteropathy, 236
Pesticide
contamination of food, discussion, 227
Phenylketonuria
association with other inherited defects, 264
diet treatment of, 234, SA353
Phosphoenolpyruvate
in muscle, in kwashiorkor, 272
Phospholipid
blood and aorta levels, relation to exercise, 55
blood levels
after egg yolk feeding, 293
effect of adiphenine derivative on, 251
effect of type of dietary protein on, 70
in swine, after feeding various fats, 108
spontaneous variations in, 100
in liver
effect of fat-mobilizing hormone on, 39
in normal and atherosclerotic aorta, 276
plasmalogen, in liver, in thiamine deficiency, 21
Phosphorus
balance, in man, after treatment with growth hormone from different species, 103
blood levels
after feeding magnesium salts, 212
in hypervitaminosis D, N158
in bone, relation to fluoride intake, 124
in milk, seasonal variations, 308
in muscle, in kwashiorkor, 272
metabolism, effect of exercise on, 42
phosphate
effect on fluoride absorption, 259
use in parenteral fluid therapy, for infantile diarrhea, 296
Physical Activity
as means of reducing weight, SA289
relation to atherosclerosis, 55, 328
relation to myocardial infarction in atherosclerosis, 221
Physical Examination
in evaluating nutritional status, SA321
Pig
growth hormone from, activity in man, 103
intestinal flora, relation to diet and age, 47
selenium utilization by, effect of calcium on, 330
Pituitary
effect of fat deficiency on, 17
source of fat-mobilizing hormone, 39
Plants
nitrogen fixation in, N191
Polycythemia
lipid deposition in, 276
Polypeptide
from algae, toxicity of, 145
Polyvinylpyrrolidone
use in determining permeability of intestine, 236
Potassium
balance, in man, after treatment with growth hormone from different species, 103
chloride
aversion to, in experimental hypertension, 57
use in management of heart disease, 35
deficiency, effect on kidney function, 242
in bone, relation to fluoride intake, 124
in muscle, in kwashiorkor, 272
K^{40}, use in measurement of body fat, 134
loss, in infantile diarrhea, 296
relation to Cs^{137} in tissues, 245
Poultry
zinc requirements for, 302
Pregnancy
effect of excessive lysine and leucine on, 342
fasting ketosis in, 187
hemoglobin and gamma globulin levels in, 149
pre-eclampsia, vitamin E treatment of, N63
Propionate
metabolism, role of cobamide coenzymes in, 85
Prostate
effect of fat deficiency on, 17
Protein
absorption, from jejunal pouch prosthesis, after gastrectomy, 300
blood, binding of vitamin A by, 344
blood levels
in normal versus alloxan diabetic rats, on atherogenic diets, 216
in protein-losing enteropathy, 236
in the hippopotamus, N31
in vitamin B_6 deficiency, 149
in zinc deficient poultry, 302
-bound iodine level
effect of adiphenine derivative on, 251
in cancer, 137
content, from food tables, accuracy of, 6
deficiency, vitamin A transport in, 344
depletion, prolonged, effect on liver, 285
dietary
effect on lysine enhancement of calcium absorption, 78
lack in maternal diet, effect on teeth of offspring, 311
relation to arginine requirements for the chick, 49
egg, amino acids in, 175
in milk, seasonal variations, 308
intake, of London busmen, 328
leaf sources, N31
liver, in rats force fed with plant proteins, 118
malnutrition
magnesium metabolism in, 38
studies by Food and Nutrition Board, SA97
metallothionein, cadmium and zinc in, 340
milk, amino acids in, 175
plasma, binding of vitamin B_{12} by, 200
storage, relation to food habits, 9
synthesis
effect of dietary cholesterol on, 113

in vitamin B_6 deficiency, 149
vegetable versus animal, effect on blood cholesterol levels, 70
Protein-Losing Enteropathy, 236
Prothrombin Time
in rats fed irradiated beef, 14, 110
Pruritis Vulvae
vitamin E treatment of, N63
Public Health
diet surveys, methodology, 6
food additives, discussion, 227
height-weight data for U. S. adults, SA193
iodination of salt, relation to incidence of thyroid cancer, 166
kwashiorkor studies in Cape Town, 324
obesity, health dangers of, SA289
relation of bone status to fluoride intake, 198
relation of water hardness to incidence of heart disease, 132
toxicity of fluorides in drinking water, 259
water supplies, toxic algae in, 145
Pyridoxic Acid
in urine
after penicillamine, 16
in pyridoxine dependency, 227
Pyridoxine
deficiency
after hydrazide therapy, 8
after penicillamine, 16
antibody formation in, 116, 315
blood transminase levels in, 120
effect on blood and liver vitamin B_{12} levels, 122
effect on liver transaminase, 22
hemoglobin and gamma globulin levels in, 149
oxalate excretion in, 88
dependency state in an infant, 227
in infant foods, N223
intake, relation to oxalate kidney stones, 306
metabolism, effect of dietary fat on, 336
role in absorption of amino acids, 16
Pyruvate
blood levels
in panthothenic acid deficiency, 156
in zinc deficiency, 111
in muscle, in kwashiorkor, 272

Radionuclides
in human milk, 308
Regeneration
cirrhotic liver, after partial hepatectomy, N139
Reproduction
efficiency, relation to dietary protein, 311
failure, in copper deficiency, 29
in cows, after I^{131} damage, 237
in mink, effect of whale liver diet on, N318
maintenance by antioxidants in absence of vitamin E, 217
Requirements
amino acids, studies by Food and Nutrition Board, SA97
arginine, for the chick, 49
calcium studies by Food and Nutrition Board, SA97
folacin, N159
magnesium
effect of dietary fats on, 92
for the rat, 178
vitamin, in different breeds of hens, SA225
vitamin B_6
for the infant, N223
relation to magnesium intake, 306
vitamin E, for the chick, 52
vitamin K, for the rat, 14
water, for sustained work, 23
zinc
for chicks, effect of dietary proteins on, 111
for poultry, 302
Research
nutritional, in the U.S.S.R., SA129
Riboflavin
effect on liver pyridoxal phosphate levels, 336
utilization, by different breeds of hens, SA225
Rice
bound niacin compounds in, 240
protein
force feeding of, kwashiorkor-like results from, 118
lysine lack in, 175

Saccharoid
blood levels, in diabetes, N254
Safflower Oil
effect on blood fat levels and atherosclerosis, 25
Salt
aversion to, in experimental hypertension, 57
effect on incidence of myocardial and renal infarcts, 331
intake, effect on bone sodium content, 152
iodized, relation of use to thyroid cancer, 166
restriction, in management of heart disease, 35
Scurvy
infantile, in the Bantu, SA257
Seasonal Variation
in blood carotenoid and vitamin E levels, 101
in nutrients, of milk, 308
in radionuclides in milk, 253
in thyroid uptake of iodine, 237
Selenium (*see* **Trace Elements**)
Self-Selection
aversion to salts, in experimental hypertension, 57
Serotonin
inhibition of thyroxine metabolism by, 174
Sesame
meal, feed for chicks, zinc deficiency from, 111
Sex Differences
in blood protein values, in the hippopotamus, N31
in height-weight data of U. S. adults, SA193
in life span of rats, 305
in mortality rate, SA289
in obesity and maturation in children, 36
in relation of body weight to growth rate, 247
in susceptibility to starvation ulcer, 151
in susceptibility to vitamin K deficiency, 110
in tendencies toward obesity, 211
in vitamins A and E blood levels, 101
Sheep
muscular dystrophy in, origins of, 41
pregnant, ketosis in, 187
white muscle disease in, selenium treatment of, 169
Shock
hemorrhagic, blood ketone levels in, N158
Sialic Acid
in metallothionein, 340
Silage
fatty acids in, utilization of, 170
Sitosterol
effect on lipid deposition in aorta, 338

Skeleton
development
in infants of diabetic mothers, 266
relation to growth rate and age, 247
Skin
changes, from niacin therapy, 325
color, in phenylketonuria, 264
condition, in evaluating nutritional status, SA321
fat, measure of obesity in children, 36
itching, in biliary obstruction, treatment with cholestyramine, 292
reactivity, to diphtheria toxin, in pyridoxine deficient guinea pigs, 315
Skin-Fold
thickness, correlation with K^{40} estimate of body fat, 134
Socioeconomic Status
relation to height and weight of U.S. adults, SA193
Sodium
balance, in man after treatment with growth hormone from different species, 103
chloride, effect on incidence of renal and myocardial infarcts, 331
depletion, effect on kidney, 242
in bone, 152
relation to fluoride intake, 124
in muscle, in kwashiorkor, 272
sulfate, aversion to, in experimental hypertension, 57
use in parenteral fluid therapy, for infantile diarrhea, 296
Solubility
teeth, in acid, relation to fluorine content, 139
Soybean
meal, feed for chicks, zinc deficiency from, 111
protein, relation to arginine requirement for the chick, 49
Special Articles
Brožek, J., Nutritional Research in the Soviet Union, Part I: Some General Aspects, 129
Brožek, J., Nutritional Research in the Soviet Union, Part II: Some Specific Aspects, 161
Gershoff, S. N., Who Is Well Nourished? 321
Hathaway, M. L., Heights and Weights of Adults in the United States, 193
Hutt, F. B., Nutrition and Genes in the Domestic Fowl, 225
Shank, R. E., Weight Reduction and Its Significance, 289
Smith, C. A., Nutritional Problems of Children, 353
Stone, D. B., Nutrition: The Forgotten Art, 1
Voris, L., Activities of the Food and Nutrition Board, 1960, 97
Walker, A. R. P., Anomalies in the Prediction of Nutritional Disease, 257
Weaver, W., Why Is Science Important? 65
Wells, O. V., Comments on the Current Food Situation, 33
Species Differences
in location of starvation ulcers, 151
in responses to dietary fats, 184
in ubiquinone levels in liver, in vitamin A deficiency, 218
Species Specificity
for growth hormone, 103
for intrinsic factor, 177
Sphingomyelin
in normal and atherosclerotic aorta, 276
Spleen
changes, in pantothenic acid deficiency, 156
copper in, after injection, 11
iron in, in experimental hemochromatosis, 83
lesions, in Whipple's disease, 230
pathology, in canine atherosclerosis, 346
selenium in, effect of calcium on, 330
weight, effect of cortisol and pyridoxine deficiency on, 22
effect of dietary cholesterol on, 184
in rats force fed with plant proteins, 118
relation to growth rate and age, 247
Sprue
non-tropical, absorption defects in, 138
Starch (*see* **Carbohydrate**)
Starvation
effect on excretion of fat-mobilizing hormone, 39
effect on liver lactic dehydrogenase, 44
effect on work output, 23
gastric ulcers from, 151
studies in the U.S.S.R., SA161
survival in, relation to body fat stores, 155
use of pemmican in, 73
Steatorrhea
from cholestyramine treatment, 292
in malabsorption syndromes, 138
I^{131} fat-pancreatin test for, 71
of diverticulosis, treatment with vitamin B_{12} and antibiotics, 299
Stomach
gastrectomy, vitamin B_{12} clearance from plasma after, 200
intrinsic factor in, in myxedema, 274
iron in, in experimental hemochromatosis, 83
ulcer
activation, from niacin therapy, 325
from starvation, 151
weight, relation to growth rate and age, 247
Stress
effect on blood cholesterol levels, 100
Strontium
absorption, effect of lysine on, 78
Sr^{90}
in British diet, 164
in milk, seasonal variations, 253
sources in diet, 245
studies by the Food and Nutrition Board, SA97
Succinate
metabolism, role of cobamide coenzymes in, 85
Sucrose (*see* **Carbohydrate**)
Sudanophilia
in aorta, relation to dietary fat and magnesium, 92
Sulfhydryl
binding of cadmium and zinc, in metallothionein, 340
Surgery
coarctation of aorta, effect on experimental atherosclerosis, 179
gastrectomy, vitamin B_{12} clearance from plasma after, 200
jejunal pouch prosthesis, after gastrectomy, 300
jejunal resection, for diverticulosis, effect on vitamin B_{12} absorption, 299
Survival
in starvation, 155
rations, use of pemmican in, 73
Swine
atherosclerosis in, effect of dietary fats on, 108

Synthesis
antibodies, in B-vitamin deficiencies, 116
ascorbic acid, intestinal, SA257
ascorbic acid and precursors, effect of drugs on, 48
ceruloplasmin, in Wilson's disease, 133
cholesterol
effect of adiphenine derivative on, 251
effect of dietary protein on, 113
effect of triparanol on, 262
in swine, effect of dietary fats on, 108
on fat-free diet, 184
fat, in meal eating versus nibbling animals, 9
fatty acid
effect of dietary protein on, 113
pattern in tissues, 275
mucopolysaccharide, in colon tissue, in vitamin A deficiency, 91
oxalate, in vitamin B_6 deficiency, 88
protein
effect of dietary cholesterol on, 113
in vitamin B_6 deficiency, 149
steroids, effect of triparanol on, 262
thiamine, by intestinal bacteria, effect of penicillin on, 60

Tea
consumption, in England, 328
Teeth
caries, role of heredity and infection, 105
development, relation to growth rate and age, 247
fluorosis, in the cow, 139
hardness, relation to fluoride intake, 124
size and caries in, relation to dietary protein in maternal diet, 311
Temperature
environmental
effect on magnesium requirement, 178
relation to catecholamine metabolism, N255
low
effect on blood cholesterol levels, 100
effect on coronary arteries, N95
Testis
effect of fat deficiency on, 17
effect of lowered environmental temperature on, N95
fat bodies in, in essential fatty acid deficiency, N127
iron in, in experimental hemochromatosis, 83
weight, relation to growth rate and age, 247
Tetracyline (*see* **Antibiotics**)
Tetraiodothyroformic Acid
blood cholesterol lowering by, 209
Thiamine
deficiency
antibody formation in, 116
effect on liver lipids, 21
in eggs, of different breeds of hens, SA225
sparing by penicillin, mechanism of action, 60
Thirst
effect on work output, 23
Thrombin
administration, to produce myocardial infarction in atherosclerosis, 221
Thrombosis
relation to fatty acid types consumed, 333
Thymine
metabolism, role of cobamide coenzymes in, 85
Thymus
weight, in essential fatty acid deficiency, N127
Thyroid
cancer, relation to iodine treatment, 166
damage, in the cow, from I^{131}, 237
goiter, research in the U.S.S.R., SA129
hormone, relation to vitamin B_{12} absorption, 274
hypothyroidism, lowering of blood cholesterol in, by tetraiodothyroformic acid, 209
Thyroidectomy
in dogs, effect of propylthiouracil on blood cholesterol, 179
Thyrotoxicosis
relation to iodine treatment of goiter, 166
Thyroxine (*see* **Hormones**)
Tissue
colon, mucopolysaccharide synthesis in, in vitamin A deficiency, 91
Tocopherols (*see* **Vitamin E**)
Toxicity
amphetamine, 202
cadmium, 340
citral, effect on intraocular pressure, N96
fluorides, for the cow, 139
fluorides, in drinking water, 259
food additives, 227
hydrazides, 8
I^{131}, for the cow, 237
iron-dextran preparations, 195
lysine and leucine, in the pregnant rat, 342
methoxychlor, N63
niacin, in treatment of hypercholesterolemia, 325
norethandrolone, for premature infants, 2
tetraiodothyroformic acid, 209
triparanol, 262
vitamin D, N158
vitamin K substitutes, for premature infants, 75
waterbloom, 145
whale liver, from vitamin A, N318
Trace Elements
cadmium, constituent of metallothionein in kidney, 340
cobalt, in cobamide coenzyme, 85
copper
deficiency, reproductive failure in, 28
depletion procedures, in treatment of Wilson's disease, 133
effect on cotton fur condition in mink, 143
effect on kidney, 11
presence in cytochrome oxidase, 80
fluoride
distribution in teeth, 124
in bovine teeth, 139
in drinking water, relation to bone status, 198
relation of intake to hardness of teeth, 124
iodine
formation from thyroxin, inhibition by serotonin, 174
I^{131} in milk, seasonal variations, 253
protein bound, in cancer, 137
radiosensitivity of cattle to I^{131}, 237
relation of treatment to thyroid cancer, 166
manganese, requirement, for different breeds of hens, SA225
selenium
effect on muscular dystrophy in guinea pigs, 20
replacement of vitamin E in diet of chicks, 52
role in muscular dystrophy in sheep, 41
treatment of white muscle disease, 169
utilization by swine, effect of calcium on, 330
zinc
constituent of metallothionein in kidney, 340

Trace Elements—(*Continued*)
deficiency, effect on hatchability and chick embryos, 148
deficiency in chicks, from soybean or sesame feeding, 111
metabolism, in postalcoholic cirrhosis, 4
requirements for poultry, 302
Triparanol
effect on blood cholesterol levels, 262
Tritium
for measure of body water in malnourished infants, 232
Tryptamine
inhibition of thyroxine metabolism by, 174
Turkey
zinc requirement for, 302

Ubiquinone
in liver, in vitamin A deficiency, 218
Ulcer
activation, from niacin therapy, 325
from starvation, 151
Urea
blood levels
effect of dietary cod liver oil on, 212
in hypercalcemic syndrome, 135
in hypervitaminosis D, N158
U.S.S.R.
nutritional research in, SA129
Utilization
calcium, effect of exercise on, 42
carotene, by cattle, 172
early versus late forages, 170
essential fatty acids, effect of trans-linoleic acid on, 62
fats, in tumor-bearing rats, 53
feeds, in the pig, effect of lactose diet on, 47
food, relation to meal eating versus nibbling, 9
magnesium, by the calf, effect of dietary constituents on, 212
nutrients, from jejunal pouch prosthesis, after gastrectomy, 300
selenium, by swine, effect of calcium on, 330
thiamine, effect on penicillin on, 60
vitamin B_{12}, relation to thyroid activity, 274
vitamins, by different breeds of hens, SA225

Vegetables
protein, effect on blood cholesterol levels, 70
source of Sr^{90}, 245
Sr^{90} content, in England, 164
vitamin B_6 content, N223
Vitamin A (*see also* **Carotene**)
association with protein, in blood transport, 344
blood levels
correlation with liver carotenoids, 172
effect of dietary cod liver oil on, 212
in man, 101
deficiency
Bitot's spots in, N350
liver ubiquinone in, 218
mucopolysaccharide synthesis in, in colon tissue, 91
evaluation of status in animals, 172
formation from carotene, by the intestine, 334
nutrition, relation to Bitot's spots, 104
relation to glaucoma, N96
relation to vitamin E, in chick diets, 52
toxicity, in whale liver, N318
Vitamin B_6 (*see* **Pyridoxine**)
Vitamin B_{12}
absorption
in diverticulosis with anemia, 299
in hypogammaglobulinemia, 138
in hypothroidism, 274
species specificity of intrinsic factor on, 177
binding by plasma proteins, 200
blood levels, in pyridoxine deficiency, 122
clearance from plasma, in leukemia and pernicious anemia, 200
cobamide coenzymes, 85
coenzyme of, chemistry, 283
deficiency, blood transaminase levels in, 122
in liver, in pyridoxine deficiency, 122
Vitamin D
effect on milk fever, 141
effect on mucopolysaccharide synthesis by colon tissue, 91
poisoning, blood studies in, N158
requirements, for different breeds of hens, SA225
Vitamin E
absorption, effect of alfalfa factor on, 189
blood and tissue levels, in vitamin E-deficient chicks, 52
blood levels
after feeding magnesium salts, 212
in man, 101
deficiency
from adding cod liver oil to diet, 212
in cystic fibrosis, 168
in the guinea pig, 20
in the monkey, erythrocyte life-span in, 154
liver ubiquinone in, 218
effect on magnesium utilization by the calf, 212
effect on mucopolysaccharide synthesis by colon tissue, 91
in alfalfa, 172
replacement by antioxidants, 217
requirement, for the chick, 52
role in muscular dystropy in sheep, 41
tocopherols, assay using chicks, 146
treatment of abortion, nephritis, heart disease, N63
Vitamin K
deficiency
from irradiated beef, 14
sex differences in susceptibility to, 110
effect on mucopolysaccharide synthesis by colon tissue, 91
substitutes, toxicity for premature infants, 75
Vitamins
history of research in the U.S.S.R., SA161

Water
algae toxins in, 145
body
in infants of diabetic mothers, 266
in malnourished infants, 232
consumption, in experimental hypertension, 57
fluoride in
relation to bone status, 198
toxicity, 259
hardness, relation to incidence of heart disease, 132
in muscle, in kwashiorkor, 272
intake, relation to heart disease, 35
restriction, effect on work output, 23
source of Sr^{90}, 245
Weight
aorta, relation to exercise, 55
body
changes during work, 23

data on U.S. adults, SA193
effect of cortisol and pyridoxine deficiency on, 22
of infants of diabetic mothers, 266
reduction
by drugs, 202
diet formulas for, SA289
programs for, SA1
relation to growth rate, 247
relation to survival in starvation, 155
Whale
liver, toxicity, N318
Wheat
bound niacin compounds in, 240
protein, force feeding of, kwashiorkor-like results from, 118
White Muscle Disease
origins of, 41
Wilson's Disease (*see* **Disease**)
Work
ability, effect of food and water restriction on, 23
Xanthurenic Acid
in urine
after hydrazides, 8
after penicillamine, 16
after tryptophan feeding, in pyridoxine dependency, 227
in vitamin B_6 deficiency, effect of magnesium on, 306
Xerophthalmia
Bitot's spots in, N350
X-Ray
studies of bone, relation of density to fluoride intake, 198

Yeast
Torula, dietary, effect on vitamin E deficiency in guinea pigs, 20

Zinc (*see* **Trace Elements**)

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CONTENTS OF VOLUME 19

No. 1, January 1961

Nutrition: The Forgotten Art, by DANIEL B. STONE 1
Use of Anabolic Hormones as Growth Stimulants 2
Zinc Metabolism in Laennec's Cirrhosis 4
Dietary Studies in Epidemiological Surveys 6
Xanthurenic Acid Excretion Resulting from Hydrazides 8
Effects of Meal Eating Versus Nibbling on Body Composition 9
Copper Toxicity in Kidneys 11
Influence of the Intestinal Flora on the Growth Rate of Mice 12
Irradiated Beef and Vitamin K 14
Absorption of Amino Acids 16
Fat Deficiency and Pituitary-Gonad Relationships 17
Vitamin E Deficiency in the Guinea Pig 20
Thiamine and Liver Lipids 21
Inhibition of Cortisol-Induced Gluconeogenesis by Pyridoxine Deficiency 22
Restriction of Food or Water and Work Output 23
Dietary Fats and Atherosclerosis in Chickens 25
Iron Enzymes in Iron Deficiency 27
Necrotizing Cardiopathy Associated with Chloride Deficiency 28
Copper Deficiency and Reproductive Failure 29
Letter to the Editor 31
American Board of Nutrition Announcement 31
Blood Serum Proteins of the Hippopotamus 31
Recent Books 32

No. 2, February 1961

Comments on the Current Food Situation, by ORIS V. WELLS 33
Dietary Management of Congestive Heart Failure, Hypertension and Angina Pectoris 35
Obesity, Growth, and Maturation in Childhood 36
Myocardial Infarction in Patients with Portal Cirrhosis 37
Magnesium Metabolism in Protein Malnutrition 38
Fat Mobilizing and Ketogenic Substances in Urine 39
Multiple Origins of Muscular Dystrophy in Lambs 41
Exercise and Calcium Utilization 42
Starvation and Lactic Dehydrogenase 44
Effects of Diet and Age on Intestinal Flora in the Pig 47
Drugs Which Induce Ascorbic Acid Synthesis in Rats 48
Substances Influencing the Arginine Requirement 49
Vitamin E-Free Diets in Chicks 52
Fat Absorption and Utilization in Tumor-Bearing Rats 53
Physical Activity and Experimental Dietary Atherosclerosis 55
Hypertension and Salt Aversion 57
Mechanism of the Thiamine-Sparing Action of Dietary Penicillin in Rats 60
Essential Fatty Acid Activity of Trans Acids 62
Letter to the Editor 63
Methoxychlor—Not a Milk Residue Problem 63

No. 3, March 1961

Why Is Science Important? by WARREN WEAVER 65
Amino Acid Excretion in Infants and Children 68
Effects of Animal Versus Vegetable Protein on Serum Lipids of Man 70
I^{131} Fat-Pancreatin Test for Steatorrhea 71
Malabsorptive Syndrome Induced by Neomycin 72
Pemmican 73
Toxicity of Vitamin K Substitutes in Premature Infants 75
Chlorpromazine Tranquilization Does Not Increase Milk Production 77
Lysine and the Absorption of Calcium 78

Relation of Pantothenic Acid to Adrenal Cortical Function ... 79
Copper in Cytochrome C Oxidase ... 80
Body Fat and Lactose Feeding ... 82
Experimental Hemochromatosis ... 83
Cobamide Coenzymes ... 85
Vitamin B_6 Deficiency and Urinary Oxalate ... 88
Beta-Glucuronidase and Atherosclerosis ... 90
Relation of Vitamin A to Mucopolysaccharide Formation ... 91
Dietary Magnesium and Experimental Atherosclerosis in Rats ... 92
Life Span and Pathology of Rats Maintained at 6°C and 30°C ... 95
Radioactive Cholesterol Labelling of Serum Lipoproteins ... 95
Citral, Vitamin A, and Glaucoma ... 96

No. 4, April 1961

Activities of the Food and Nutrition Board, 1960, by LeRoy Voris ... 97
Spontaneous Variations in Serum Lipids and Cholesterol ... 100
Levels of Vitamins A and E in Human Blood ... 101
Growth Hormone Studies in Man ... 103
Bitot's Spots ... 104
Hereditary and Infectious Components of Tooth Decay ... 105
Atherosclerosis in Miniature Pigs ... 108
Sex Differences in Susceptibility to Vitamin K Deficiency ... 110
Zinc Deficiency in Chicks ... 111
Dietary Protein and Acetate-1-C^{14} Incorporation ... 113
Fatty Acid Uptake by Skeletal Muscle ... 115
Antibody Formation by Vitamin Deficient Rats ... 116
Linoleic Ester and Cholesterol ... 117
Kwashiorkor and Plant Proteins ... 118
Pyridoxine and Transaminases ... 120
Vitamin B_6 and B_{12} Relationships ... 122
Injury, Growth, and Nitrogen Balance ... 123
Fluoride Content of Skeletal Tissues ... 124
Liver Enzymes in Glycogen Storage Disease ... 127
Fatty Acid Chain Length and Essential Fatty Acid Deficiency ... 127
Recent Books ... 128

No. 5, May 1961

Nutritional Research in the Soviet Union. Part I: Some General Aspects, by Josef Brožek ... 129
Relation Between Hardness of Water and Cardiovascular Disease ... 132
Copper Elimination Treatment of Hepatolenticular Degeneration ... 133
Total Body Fat Determined by Potassium-40 Content ... 134
Prognosis in the Infantile Hypercalcemic Syndrome ... 135
Unesterified Fatty Acid Levels in Neoplastic Disease ... 137
Rare Malabsorption Syndromes ... 138
Dental Effects of Fluorine Ingested by Bovines ... 139
Vitamin D and Milk Fever Prevention ... 141
Fish and Unavailable Iron ... 143
Toxic Waterbloom (Algae) ... 145
Assay and Biopotency of Alpha-Tocopherols ... 146
Malformations in Zinc-Deficient Chick Embryos ... 148
Blood Alterations in Vitamin B_6-Deficient, Pregnant Rats ... 149
Starvation Ulcers ... 151
Bone Sodium ... 152
Vitamin E and Erythrocyte Life-Span ... 154
Starvation and Body Composition ... 155
Pantothenic Acid Deficiency in Guinea Pigs ... 156
Blood Ketones During Hemorrhagic Shock ... 158

Hypervitaminosis D ... 158
The Daily Dose of Folacin ... 159
Letter to the Editor ... 160
Recent Book ... 160

No. 6, June 1961

Nutritional Research in the Soviet Union. Part II: Some Specific Aspects, by JOSEF BROŽEK ... 161
Strontium-90 in the British Diet ... 164
Relation of Endemic Goiter to Thyroid Carcinoma and Thyrotoxicosis ... 166
Tocopherol Deficiency in Infancy and Childhood ... 168
Selenium, Transaminase and White Muscle Disease in Lambs ... 169
Major Factors Influencing Forage Value in Dairy Cattle ... 170
Evaluation of Vitamin A Status of Animals ... 172
Hormonal Effects of Tryptophan Metabolism ... 174
Milk and Egg Supplementation of Rice ... 175
Intrinsic Factor and Absorption of Vitamin B_{12} ... 177
Magnesium Requirement of Rats ... 178
Interrelation of Aortic and Coronary Arterial Hypertension with Dietary-Induced Hypercholesteremia on Atherosclerosis in Dogs ... 179
Hypothalamic Stimulation and Feeding Behavior ... 182
Endogenous Formation of Cholesterol on a Fat-Free Diet ... 184
Glutamic Acid and Growth ... 186
Fasting Ketosis in Pregnant Animals ... 187
A Fat Soluble Material in Alfalfa Which Reduces Availability of Dietary Tocopherol ... 189
Heart Disease in Algeria ... 191
Nitrogen Fixation by Nonleguminous Plants ... 191
Letter to the Editor ... 192
Recent Books ... 192

No. 7, July 1961

Heights and Weights of Adults in the United States, by MILICENT L. HATHAWAY ... 193
Iron Deficiency in Prematurely Born Infants ... 195
Bone Density and Fluoride Ingestion ... 198
Clearance of Vitamin B_{12} from Plasma ... 200
The Drug-Management-of-Obesity Controversy ... 202
Exercise and Blood Ammonia Levels in Man ... 206
Clearance of Intravenous Fat ... 207
Reduction of Serum Cholesterol by Tetraiodothyroformic Acid ... 209
Obesity in Childhood and Adulthood ... 211
Effects of Vitamin E and Cod Liver Oil on Magnesium-Deficient Calves ... 212
Eating Habits and Atherosclerosis ... 213
Atherogenic Diets in Diabetics ... 216
Antioxidant Replacements for Vitamin E ... 217
Ubiquinone in Vitamin A Deficiency ... 218
Physical Activity and Myocardial Infarction ... 221
A Hypothalamic Fat Center ... 222
Vitamin B_6 Content of Infant Foods ... 223
Recent Books ... 224

No. 8, August 1961

Nutrition and Genes in the Domestic Fowl, by F. B. HUTT ... 225
Food Additives ... 227
Vitamin B_6-Dependency State in Infants ... 229
Electron Microscopic Studies in Whipple's Disease ... 230
Total Body Water in Malnourished Infants ... 232
Results of Treatment in Phenylketonuria ... 234
Protein Losing Enteropathy ... 236

Radio Toxicity of Iodine^{131} in Dairy Cattle 237
Bound Niacin 240
Potassium Deficiency and the Kidney 242
Long-Term Feeding of Fats and Atherosclerosis in Chicks 244
Dietary Components and Accumulation of Radionuclides in the Body 245
Accelerated Growth of Rats 247
Regulation of Cholesterol Metabolism with Thyroxine Analogues 250
Lowering of Serum Cholesterol by an Adephenine Derivative 251
Radionuclides, Calcium, and Potassium in Milk 253
Blood Saccharoids 254
Environmental Temperatures and Urinary Metabolites of Noradrenaline 255
Recent Books 256

No. 9, September 1961

Anomalies in the Prediction of Nutritional Disease, by ALEXANDER R. P. WALKER 257
Metabolism of Fluorides 259
Reduction of Serum Cholesterol Concentration by Triparanol 262
Phenylketonuria 264
Body Composition of Infants Born to Diabetic Mothers 266
Alcoholic Cardiomyopathy 268
Calcium Metabolism in Osteoporosis 269
Muscle Composition in Kwashiorkor 272
Relationship Between Thyroid Hormone and Vitamin B_{12} 274
The Effect of Linoleic Acid on Depot Fat 275
Fatty Acid Analysis of Lipid Fractions 276
Ascorbic Acid Requirement of Guinea Pigs 278
Nicotinic Acid and Starvation Hypercholesteremia 280
Anemia, Polycythemia, and Lipid Deposition 281
Coenzyme B_{12} in Liver 283
Prolonged Protein Depletion 285
The Effect of Insulin on Hepatic Glucose Output in Man 286
Catalog of Films and Filmstrips on Food and Nutrition 287
Announcement 288

No. 10, October 1961

Weight Reduction and Its Significance, by ROBERT E. SHANK 289
Effects of Cholestyramine, A Bile Acid Sequestering Exchange Resin 292
Dietary Cholesterol and Serum Lipids in Man 293
Parenteral Fluid Therapy in Infantile Diarrhea 296
Diverticulosis of the Jejunum with Macrocytic Anemia and Steatorrhea 299
Absorption and Utilization of Nutrients in Man After Total Gastrectomy 300
Zinc Requirements of Poultry 302
Nutrition, Health, and Longevity 305
Renal Stones, Magnesium, and Vitamin B_6 in Rats 306
Composition of Milk 308
Reduction of Serum and Liver Cholesterol with Pectin 309
Nutrition, Tooth Size, and Caries Susceptibility 311
Fatty Acid Chain Length and Metabolic Rate 314
Impaired Antibody Production in Pyridoxine Deficient Guinea Pigs 315
Antibiotic-Containing Foods and Animal Experimentation 317
Vitamin A Toxicity in Whale Liver 318
Liver Regeneration After Partial Hepatectomy in Cirrhotic Rats 319
Recent Books 320

No. 11, November 1961

Who Is Well Nourished? by STANLEY N. GERSHOFF 321
Kwashiorkor in Cape Town 324

Treatment of Hypercholesterolemia with Nicotinic Acid 325
Diet and Atherosclerosis in London Busmen 328
Effect of Excessive Calcium on Selenium 75 Utilization in Swine 330
Sodium Chloride and Myocardial Infarcts in Rats 331
Dietary Fats and Atherosclerosis and Thrombosis in the Rat 333
Intestinal Conversion of C^{14}-Carotene to Vitamin A 334
Fatty Acids and Riboflavin in the Metabolism of Vitamin B_6 336
Experimental Atherosclerosis in Dogs with Aortic Grafts 338
Cadmium, A Possible Dietary Essential 340
Effect of Excessive Lysine and Leucine Intakes on the Pregnant Rat 342
Vitamin A and Proteins in Blood and Liver 344
Pathologic and Physiologic Studies in Canine Atherosclerosis 346
Trivalent Chromium and Glucose Tolerance 348
Letter to the Editor 350
Chemicals in Food 351
Dietary Treatment of Familial Mediterranean Fever 352
Recent Books 352

No. 12, December 1961

Nutritional Problems of Children, by CLEMENT A. SMITH 353
Author Index 357
Subject Index 365

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